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THE PLANT DISEASE REPORTER

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Division of Mycology and Disease Survey

BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING

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The Plant Disease Reporter is issued as a service to plant pathologists throughout the United States. It contains reports, summaries, observations, and comments submitted voluntarily by qualified observers. These reports often are in the form of suggestions, queries, and opinions, frequently purely tentative, offered for consideration or discussion rather than as matters of established fact. In accepting and publishing this material the Division of Mycology and Disease Survey serves merely as an informational clearing house. It does not assume responsibility for the subject matter.

THE PLANT DISEASE REPORTER

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THE PLANT DISEASE SURVEY
DIVISION OF MYCOLOGY AND DISEASE SURVEY

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SUGARBEET CURLY-TOP VIRUS AND CURLY-TOP DISEASE IN ILLINOIS AND THEIR RELATION TO HORSERADISH BRITTLE ROOT

H. H. Thornberry and R. M. Takeshita

Sugarbeet curly-top virus (Ruga verrucosans Carsner & Bennett, 1943) and sugarbeet curly-top disease are herein reported to occur in Illinois; the first announcement of the occurrence of the virus and the disease in Illinois based upon valid evidence. Since the virus can attack many other crops and plants, the disease is of potential importance in the State and in the region.

The evidence supporting the announcement consists of the following findings: The leafhoppers tentatively identified as Circulifer tenellus (Baker) were collected in the East St. Louis area from horseradish on July 1, 1953 (see acknowledgment 1, at end) and identified July 28, 1953 (see acknowledgment 2). The presence of the virus was established (see acknowledgment 3) in specimens of the leafhopper vector collected August 5 and 14, 1953 from horseradish in Madison County, Illinois. Sugarbeet plants suspected of having the curly-top disease were detected on August 9, 1953 just north of Chicago in Cook County. Some of these plants were found to contain the virus (see acknowledgment 3).

The following comments from Dr. N. J. Giddings are pertinent to the identity of the virus in leafhopper and sugarbeet specimens collected in Illinois:

1. Letter of August 31, 1953 relative to leafhoppers collected August 5 (first shipment) and August 14 (second shipment): "It will be of interest to you to know that I have recovered curly top from the first shipment and from the second shipment. These tests were very carefully handled and I am sure that the results are accurate."
2. Letter of September 1, 1953: "It is definitely established that you have curly top viruliferous insects in your general area."
3. Letter of September 23, 1953: "I have confirmatory evidence that one of the sugarbeet roots which you sent is diseased with curly top. One of the others looked rather suspicious and it is possible that further tests may give evidence of virus, but we have obtained no such evidence from that or any of the other 3 plants as yet. It is now certain that you have curly top, both in the beet and in the leafhoppers, from that area. This is the first definite proof of the existence of the disease there."

The leafhopper, C. tenellus, has been collected (2) and identified (1) previously in Illinois. DeLong (1) classified the leafhopper at that time under the name of Norvellina tenella (Baker) and states that, "This is a widely distributed and an important economic species. Its occurrence in Illinois is both unusual and interesting. Several years ago it was found on its native food plant, the sea purslane, Sesuvium portucastrum, along the Atlantic Coast on sandy beaches, but as far as is known these beach plants are not found in Illinois. It has been taken in abundance from a planting of horseradish in Illinois, where it caused a diseased condition similar to curly top of sugar beets." In the same publication and under Illinois Records (1)--"Collinsville: on horseradish, October 6, 1936, K. J. Kadow, 58♂, 34♀; on horse-radish, July 14, 1938, L. H. Shropshire, 1♀." These comments and records establish the occurrence of this leafhopper on horseradish in Illinois but do not prove that the curly-top virus induced the disease on horseradish.

The relation of curly-top virus to the horseradish brittle root disease in Illinois has been suspected since the disease was first reported and described by Kadow and Anderson in 1936 (2). These authors also suggested but gave no proof that curly-top virus was the cause of the brittle root disease. In a later publication (3) they describe the disease further and state that "Brittle root is evidently caused by the curly-top virus (page 554, Fig. 14). . . . Wherever symptoms of curly top are visible above ground, root symptoms are always clearly defined." (page 555). The identification was based largely on the similarity between the symptoms of the brittle root disease observed in Illinois and those reported for curly-top of horseradish as described and illustrated by Severin (4) in California.

The horseradish brittle root disease, as recognized by growers in Illinois, appears to be related to a complex of causes one of which may be the sugarbeet curly-top virus. The disease in the East St. Louis area this year was apparently affected by the severe drought and high temperatures during the growing season. An estimated crop loss of approximately 80 percent for the East St. Louis area consisting of Madison and St. Clair Counties has been

attributed to the disease this year. In Cook County, the other primary horseradish growing area of the State, the disease has been of little consequence although it has been observed on a few plants this growing season. Only a few leafhoppers were detected in horseradish and sugarbeet fields in Cook County throughout this season.

On July 14, the leafhoppers were found on experimental horseradish plants in Champaign County (see acknowledgment 1 and 2). Throughout the season they have been collected from horseradish plants in this area. The brittle root disease of horseradish was detected in Champaign County last year for the first time. Experimental plants have been grown in the county continually since 1949 and intermittently since 1938 without recognizable cases of the disease.

On October 1, sugarbeet plants with typical symptoms of curly top were observed in Cook County. In some fields approximately 5 percent of the plants showed typical symptoms. In other fields plants with typical symptoms were not observed. However, many plants showed curled young leaves without vein clearing or enlargement. The vector was present in these fields.

On October 2, cucumber plants (variety unknown) growing adjacent to a horseradish planting in Madison County had symptoms suggestive of curly top on cucumber as described and illustrated by Severin and Henderson (5). Other curly-top susceptible species of plants growing in or adjacent to horseradish fields in Madison County did not reveal recognizable symptoms of curly top. Failure to recognize symptoms in these plants may have been due to unfamiliarity with symptoms of the disease and to the effect of the drought upon the growth of the vegetation in the area.

On October 21, horseradish plants in Peoria County were observed to have brittle root comparable in amount to that in Cook County. The curly-top vector was also present. Two farmers, the only known horseradish growers in the area, claim that the disease has been present for only the past two years. They also claim that they use their own sets and have not obtained sets from plants grown elsewhere. On the strength of these claims, it would appear that the disease has not been introduced into this area from other areas by means of set roots carrying the causal agent. The origin of the disease in the area is not known.

The distribution of the sugarbeet curly-top virus vector was probably throughout Illinois in 1953 since known occurrence of the leafhopper included widely separated areas of the southwestern, central, east central, and northeastern portions of the State. There was a distinct relationship between leafhopper population and prevalence of the horseradish brittle root disease. Wherever brittle root occurred, the leafhopper was also present. However, a causal relation of the curly-top virus vector to the brittle root disease has not been established. Experiments are in progress to ascertain whether the curly-top virus is responsible for a part or the whole of the brittle root disease as it is presently recognized by growers.

Acknowledgments

Contributions to this report by the following individuals are gladly acknowledged:

1. Mr. H. B. Petty, Extension Specialist in Entomology, Illinois Natural History Survey, Urbana, Illinois, for collecting leafhoppers from horseradish plants and tentatively identifying them as Circulifer tenellus.
2. Dr. M. W. Sanderson, Associate Taxonomist in Entomology, Illinois Natural History Survey, Urbana, Illinois, for the final identification of leafhoppers as Circulifer tenellus (Baker).
3. Dr. N. J. Giddings, Senior Pathologist, U. S. Department of Agriculture Sugar Plant Investigations Laboratory, Riverside, California, for the identification of the curly-top virus in the leafhoppers and diseased sugarbeet plants; and for cooperation in previous years in unsuccessful attempts to obtain the curly-top virus from horseradish plants showing symptoms of brittle root and sugarbeet plants suspected of having curly-top virus.

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their control. Illinois Agr. Exp. Sta. Bull. 469: 531-583.
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UNIVERSITY OF ILLINOIS, URBANA

A MOSAIC OF SWEETPOTATO IN PLANT BEDS AND FIELDS IN GEORGIA[1]

H. I. Borders[2] and T. J. Ratcliff[3]

Sweetpotatoes are grown in Georgia not only for the roots which are marketed and used on the farms, but also for "seed" sweetpotatoes and for sweetpotato plants. Georgia State law provides for mandatory inspection of sweetpotatoes and no "seed" sweetpotatoes, plants, or vine cuttings may be sold, given away, or otherwise distributed unless certified by inspectors of the Georgia Department of Entomology as complying with Georgia regulations.

In 1945 inspectors of the Georgia Department of Entomology found off-type plants of Bunch Porto Rico sweetpotato on a farm in Baldwin County, Georgia. These "off-type" plants had symptoms resembling a mosaic type of virus infection and there was no yield of roots from the diseased plants. These symptoms had not been previously reported in Georgia. As only a few affected plants were found, they were rogued and notations were made on the inspection cards to make subsequent follow-up inspections of the area. The following year, 1946, a greatly increased number of mosaic-infected plants were found on this farm in plantings from the same stock, and the entire planting stock of this grower was condemned and destroyed. No more plants with mosaic symptoms were found by the inspectors until the late summer and early fall inspections of sweetpotato fields in August, September, and October, 1952.

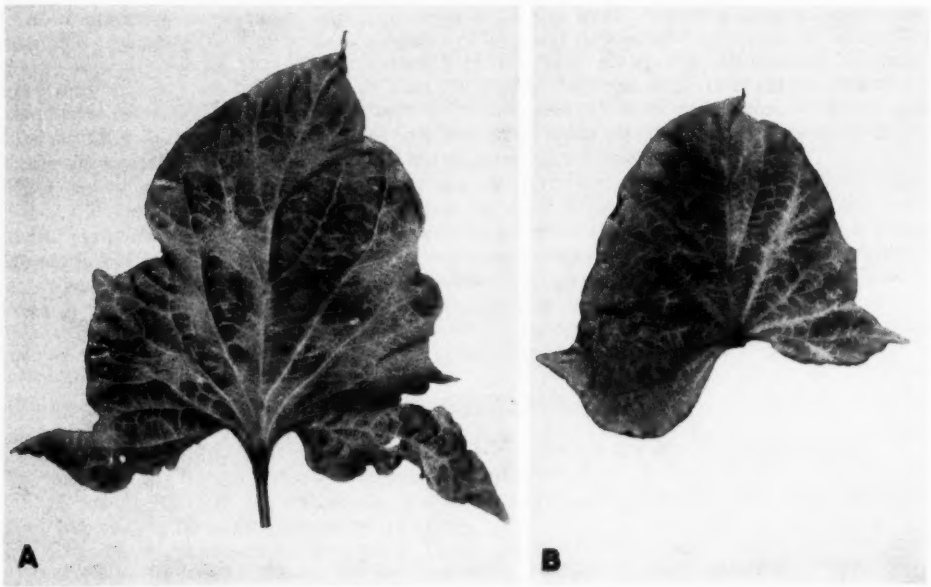

FIGURE 1.--Mosaic symptoms in Bunch Porto Rico sweetpotato. A, leaf showing mottling, blistering, and distortion in outline; B, leaf showing yellowing of many small veins similar to that caused by feathery mottle virus.

In early fall of 1952 the inspectors found a number of sweetpotato fields in which almost 100 percent of the vines were infected by what appeared to be a virus. The diseased plants showed a mottling of the older leaves with raised blister-like dark green areas which gave the

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[1] Contribution of the Department of Plant Pathology, Georgia Coastal Plain Experiment Station. Published with approval of the Resident Director as Journal Series Paper Number 16.

[2] Pathologist, Bureau of Plant Industry, Soils, and Agricultural Engineering, United States Department of Agriculture.

[3] Chief Inspector, Georgia Department of Entomology.

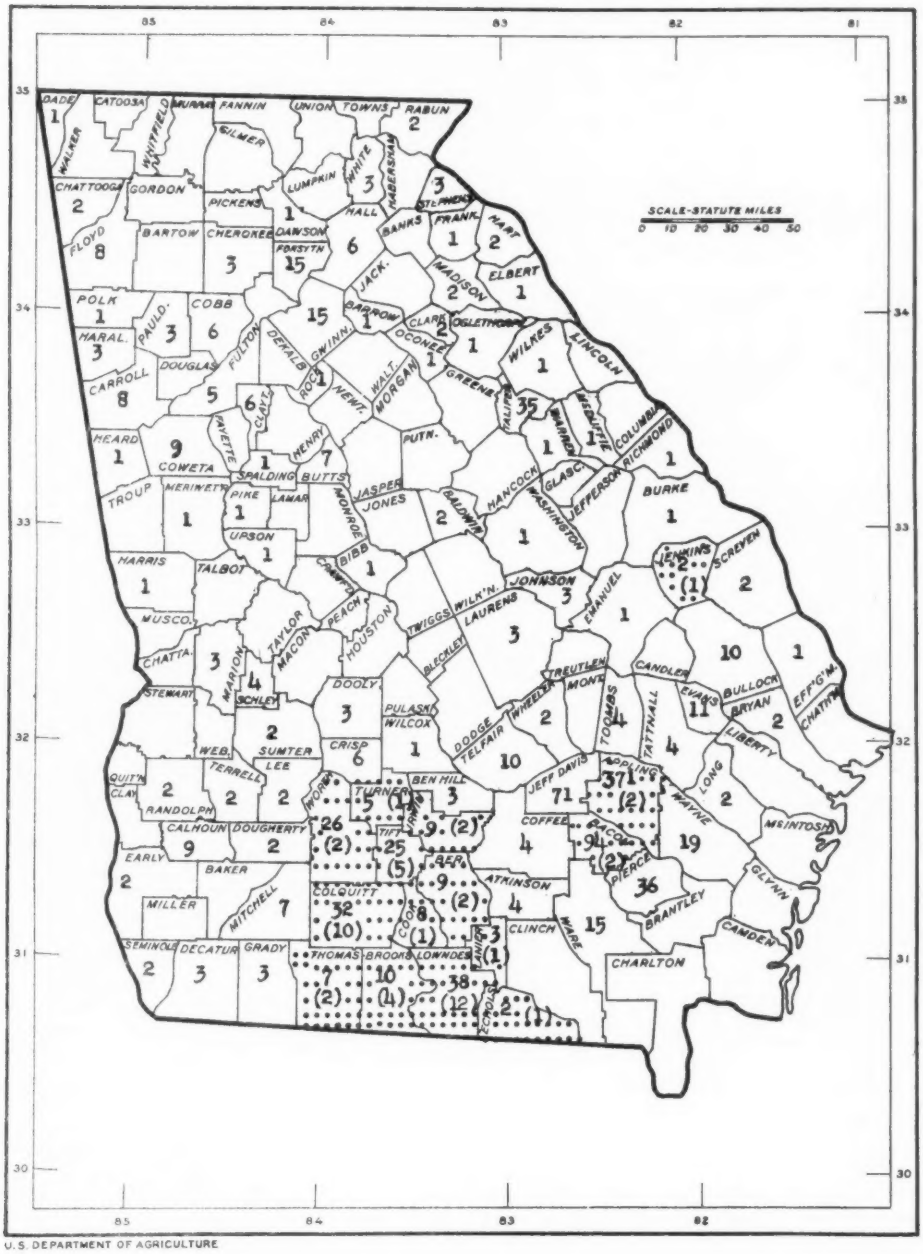

FIGURE 2. Prevalence of sweetpotato mosaic in Georgia as indicated by findings of Georgia Department of Entomology in August and September, 1953.

Dots indicate counties in which mosaic was found during inspections in August and September, 1953.

Numbers in parentheses indicate inspections in which mosaic was found. Numbers without parentheses show total inspections in each county.

leaves a somewhat wrinkled appearance. Such leaves frequently were distorted in outline (Fig. 1A). Many of the small veins on some young leaves showed a yellowing (Fig. 1B) very similar to that caused by the feathery mottle virus (Flavimacula ipomoeae) described by Doolittle and Harter (1). However, the mottling and blistering of the leaves were the predominant symptoms and appeared to be much more common than in the plants observed by Doolittle and Harter. The internodes of diseased plants were shortened and only a few very small fleshy roots were produced, whereas the uninfected plants in the Georgia fields produced normal yields. Official inspections by the Georgia Department of Entomology during the late summer and fall of 1953 resulted in the finding of this disease in sweetpotato fields in 15 counties in the coastal plain of Georgia (Fig. 2).

Since the disease was of definite importance, its investigation was undertaken by the senior author at the Vegetable Seedling Disease Laboratory, Bureau of Plant Industry, Soils, and Agricultural Engineering, United States Department of Agriculture, at the Georgia Coastal Plain Experiment Station, Tifton, Georgia. Samples of diseased plants were collected and work was carried on in the greenhouse during the winter of 1952-53 and in the field during the summer of 1953.

Greenhouse Experiments

In September and October, 1952, diseased sweetpotatoes and vine cuttings were planted in pots and beds in the greenhouse. Healthy sweetpotatoes were also obtained and planted for use as a source of vine cuttings and sprouts. One source of healthy material was sent by S. P. Doolittle from a plant at the Plant Industry Station, Beltsville, Maryland, (No. 029878). This plant was from healthy stock of the variety on which the feathery mottle virus was first noted (1). Another plant, of the variety Cliett Bunch Porto Rico, was furnished by the Georgia Department of Entomology.

Beginning in November, 1952, a number of inoculations were made with extracted juices (1:5 and 1:10 dilutions with water) of leaves, stems, and roots of diseased plants. Leaf inoculations were made by the usual rubbing method using carborundum powder. Roots of cuttings were inoculated by crushing or cutting the roots immersed in an extract of the diseased plant tissues. Fifty each of the above types of inoculations were made. All the inoculated plants were held for 60 to 180 days but no symptoms were observed on any of them.

However, the virus was readily transmitted when healthy cions were tip-grafted to diseased plants of Cliett Bunch Porto Rico and No. 029878 sweetpotato. In all cases of core-grafting of diseased root cores to healthy roots, virus transmission took place. Within 9 to 11 days after insertion of the cion in the infected stock, virus transmission to tip-grafts was evident in both the bunch and the vining types of sweetpotatoes by the appearance of mosaic patterns on the leaves of the cion and by puckering and distortion of its leaves. Healthy sweetpotato roots core-grafted with cores from roots of virus-infected plants produce sprouts which show the typical mottling and puckered-leaf symptoms of the virus, together with the shortened internodes and somewhat "feathery-edge" leaves. The above grafts were replicated 25 times.

In addition, 20 to 25 young plants of various species were inoculated by means of leaf inoculations, hypodermic injections, placing ground diseased tissue in slits in stems, scarifying the outer tissue of plants, or slicing off a thin longitudinal section on the stem of the plant inoculated and fastening it to a similarly treated stem of a diseased sweetpotato plant as though one were making a modified "approach graft". The plants used in this test were tomato, Lycopersicon esculentum Mill., var. Rutgers; tobacco, Nicotiana tabacum, L., var. Hicks; bean, Phaseolus vulgaris L., var. Tendergreen; pepper, Capsicum frutescens L., var. World Beater; cucumber, Cucumis sativus L., var. Straight Eight; squash, Cucurbita pepo L., var. Yellow Summer Crookneck; cabbage, Brassica oleracea L., var. Charleston Wakefield; morning glory, Ipomoea purpurea (L.) Roth. var. Crimson Rambler; and I. tricolor Cav., var. Heavenly Blue; wild morning glory, Ipomoea sp.; amaryllis, Amaryllis sp.; and poinsettia, Euphorbia pulcherrima Willd. In none of these tests was there any evidence of transmission of the virus.

Sweetpotato Field Plot Experiments

Field plot experiments were started in June, 1953, in a field on a farm in one of the southern tier of south Georgia counties. This field was selected because sweetpotatoes grown there in 1952 apparently had all been infected with the virus.

One of the objectives of the field tests was to find whether planting alternating rows of healthy and virus-infected sweetpotato plants would result in disease incidence in the healthy

plants. In this experiment two rows of healthy plants of the variety Cliett Bunch Porto Rico were planted alternately with single rows of mosaic plants. By the end of the season nearly 100 percent of the plants from the healthy stock showed typical mosaic symptoms.

In another portion of the field plot, healthy sweetpotato plants were planted under field cages of 32 x 32 mesh Lumite screen in order to control possible insect vectors. The caged plants were alternated with healthy plants grown in the open. Also there was set up an insecticide spray experiment in which rows of healthy sweetpotato plants grown in the open received regular applications of malathion spray at 7-day intervals at the rate of 175 to-200 gallons per acre containing 2 pounds of malathion (actual) per 100 gallons. Leaf hopper infestation of the uncaged, unsprayed plants was heavy whereas plants in the cages and those protected by the malathion spray were very nearly free of insect injury.

These experiments were terminated during the latter part of September, 1953. No virus symptoms could be found on any of the sweetpotato plants grown under the cages nor were there any virus-infected plants in the portion of the test plot which had received regular weekly spray applications of malathion. The healthy plants grown in alternating rows of diseased and healthy plants without the protection of cages or spray applications all became infected by the virus as evidenced by the typical mosaic pattern described previously.

Yield data from field plot experiments as well as observation of yields from commercial fields show that healthy vines produce approximately 27 percent larger yield than do vines that become infected during the growing season. There was no yield, other than a few small culls, from virus-infected draws or vine cuttings.

The results of this one season's experiments indicate that the virus is not soil-borne but is carried by an insect vector.

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A SIMPLE APHID CAGE FOR VIRUS TRANSMISSION STUDIES

Paul M. Eide[1] and D. M. McLean[2]

In certain aphid transmission studies with viruses it frequently becomes necessary to make a great number of single or multiple aphid group transfers and to confine these insects on test plants for varying periods of time. A number of different types of cages for this purpose and methods of transferring aphids have been described and used successfully by other workers[3]. In strawberry virus plant indexing studies at the Northwestern Washington Experiment Station a simple cage for confining infective strawberry aphids on leaves of test plants has been used very efficiently for several years. The cages may be used repeatedly, are light in weight, and are very easily manipulated. It is possible to index 100 strawberry test plants in a day by confining 3 to 5 aphids per cage. These cages are also being used successfully and conveniently in transmission studies of table beet viruses. Because of the possible use other workers might find for these cages their simple construction is here described and one is illustrated (Fig. 1).

The necessary materials are commonly available and the only tools needed are No. 6 and No. 10 cork borers and a sharp knife or razor blade. The supporting arms of the clamp are cut from 3/32-inch balsa wood sheets obtainable at handicraft supply shops. The circles of cork that form the cage and make close contact with the leaf of the test plant, and the hinge supports, are cut from 3/32-cork gasket material obtainable from automotive supply shops. The cloth parts of the cage and the hinge of the clamp are organdy. Transparent covers of the cages are made of thin celluloid sheet supported on short cylinders of 52/52-mesh lumite screen. This celluloid is obtainable from handicraft shops. Assembled parts are held together with acetate cement and the clamp is held closed with a No. 8 rubber band.

The arms of the clamp are 2 1/2 by 3/4 inches with holes made with a No. 6 cork borer 1/4 inch from the ends. The cork circles are made by first cutting a hole in the sheet cork with a No. 6 borer and then cutting out the ring by a concentric cut with a No. 10 borer. Circles of organdy and of celluloid are cut with the No. 10 borer. Hinges are made by cementing a 3/4 by 1/2 inch piece of organdy to one side of a 3/4 by 1/2 inch piece of the cork, cutting the cork almost through at the midline of the long dimension, and then folding the assembly at the cut line with the organdy inside the fold (see illustration, Fig. 1).

The non-transparent cage is assembled as follows: A circle of organdy is cemented concentrically over the hole in one arm and a ring of cork is cemented concentrically over the organdy. Another circle of cork is cemented concentrically around the hole in the second arm. The hinge is then cemented about 3/8 inch from the ends of the two arms opposite the holes, with the fold of the organdy toward the near ends of the arms, and with the cork circles neatly coincident. The assembly is held together by a rubber band about it at the midpoint, until the cement has hardened. (Fig. 1, A).

A cage with transparent top for observation of caged insects is assembled as follows: Cork rings are cemented around the holes in both arms, without organdy under either, the hinge is attached, and the assembly is held by a rubber band as described above. A strip of lumite screen 1 5/8 by 3/8 inch is bent into a short cylinder of the same diameter as the holes. This cylinder is cemented into the hole with one edge flush with exposed surface of the cork ring and the other edge extending 3/16 inch above the outer surface of the arm. The circle of celluloid film is then cemented across the top of the cylinder and projecting above the arm. Care must taken to avoid closing the exposed mesh of the lumite screen with misplaced cement. (Fig. 1, B).

The dimensions given are for cages that best meet the requirements of the authors, but size can be varied to suit other conditions or preferences.

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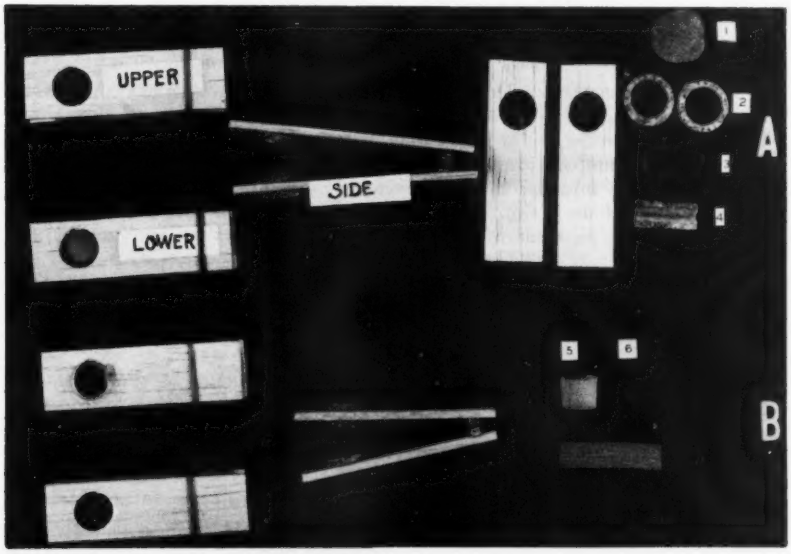
[3] Smith, F. F., Journal of Economic Entomology 24: 914. 1931; Smith, K. M., Annals of Applied Biology 19: 308. 1932; Mellor, F. C. and R. E. Fitzpatrick, Canadian Journal of Botany 29: 412. 1951; Lochner, E. H. W., Science Bulletin No. 333 (Entomology Series No. 36) Union of South Africa, Department of Agriculture. 1952.



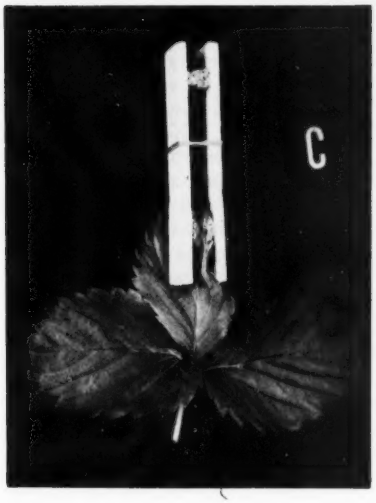


FIGURE 1. Simple aphid cage for virus inoculation studies: A -- 1, Circle of organdy; 2, Cork collars; 3, Rubber band; 4, Cork hinge. B -- 5, Lumite insect screen; 6, Circle of celluloid. C -- Cage in position on strawberry leaf.

Aphids may be transferred from the source plant with a moistened camels-hair brush and placed directly in the cage which is then clamped over a leaf of the test plant, or aphids may be placed directly on the test plant and the cage placed over them. The latter method is used most successfully with the strawberry aphid while the former is used with the green peach aphid. Up to 20 apterae have been confined successfully in single cages (Fig. 1, C).

Where it becomes desirable to expose aphids to pre-infection or post-infection fastings it is only necessary to place them in the cage and place a piece of paper between the collars confining the aphids. Following the fasting period the paper is removed and the cage is clamped in place on a leaf of the test plant.

Many aphids normally seek the lower leaf surfaces for feeding courts. In inoculation and transmission studies aphids usually are placed in the cages confined to the lower leaf surfaces.

NORTHWESTERN WASHINGTON EXPERIMENT STATION, MOUNT VERNON, AND U. S. BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING

DISCOVERY OF EXOCORTIS IN FLORIDA CITRUS[1]

L. C. Knorr, E. P. DuCharme, and J. N. Busby

The trifoliate orange, Poncirus trifoliata (Linn.) Raf., possesses important advantages to commend it as a rootstock for citrus. Besides producing fruits of high internal quality, it is highly resistant to cold, citrus nematodes (Tylenchulus semipenetrans Cobb), foot rot, and water damage. It is also tolerant or immune to the virus disease tristeza, and for this reason alone is attracting considerable interest in areas invaded by tristeza, especially in localities with heavy or wet soils where other tristeza-tolerant rootstocks like rough lemon and sweet orange grow poorly or succumb to foot rot.

FIGURE 1. Exocortis scaling of the bark in a trifoliate-orange rootstock. The sweet orange portion of the tree remains unaffected. (Photograph by courtesy of Dr. I. W. Wander)

These advantages, however, are offset by the susceptibility of this rootstock to exocortis, a disease that causes a scaling of the butts (Fig. 1) and a stunting of the tops. Exocortis or "scaly butt" may at times assume considerable importance, as for instance in Argentina, where groves killed by tristeza and subsequently replanted with trifoliate rootstocks have had to be replanted a second time because of exocortis. This happened notably in four groves in the Litoral where 16,500 trees on trifoliata were pulled recently because of this disease.

Exocortis has been reported previously from citrus areas throughout the world. The first published account appeared in 1948 when Fawcett and Klotz (2) described and named the disease and recorded its presence to a limited extent in California groves. Earlier, in 1938, it was observed by Camp[2] in Louisiana where it had been a problem for many years. Brotas[3] mentions

[1] Florida Agricultural Experiment Station Journal Series, No. 241.

[2] A. F., Camp, Citrus Experiment Station, Lake Alfred, Florida, in conversation with LCK, July 25, 1951.

[3] Lucia Koch de Brotas, Uruguayan Min. Gan. y Agr., in conversation with LCK, December 13, 1950.

its occurrence in Uruguay, Knorr, DuCharme, and Banfi (3) in Argentina, Benton and associates (1) in Australia, and McClean (4) in South Africa.

A scaling of trifoliate butts was first reported in Florida during the summer of 1952. Inspectors of the State Plant Board noted the abnormality in 25-year-old sweet orange trees on trifoliate rootstocks growing at Winter Garden. Subsequent cases of trifoliate scaling were reported from Apopka in 12- and 20-year-old sweet orange trees, at Jacksonville, in a 20-year-old navel orange tree, and at Dunedin in 30-year-old seedy grapefruit trees. An examination of these cases and a comparison of symptoms with those observed in Louisiana, Argentina, and Uruguay (3), indicate that they are affected with the exocortis disease.

Despite the recency of this report of exocortis in Florida, there are indications that the causal agent (considered by Benton et al. (1) to be a bud-transmissible virus) has been in Florida for some time. An instance is known of Florida budwood that was sent to Louisiana, where it was budded into trifoliate seedlings. Subsequently these trees developed symptoms of exocortis. A similar situation was encountered recently by the first author at Salto, Uruguay. Approximately 100 trifoliate-rooted trees were found severely stunted and attacked by exocortis scaling. Twenty years earlier, the grove owner made these trees by budding trifoliate seedlings with budwood of Duncan grapefruit from Florida. At that time other trifoliate seedlings of the same lot were budded to Thompson, Foster, and Marsh Seedless grapefruit buds also from Florida, but today none of the trees of these latter budwood sources shows stunting or scaling.

That insects are not involved in the dissemination of exocortis, at least not under Uruguayan conditions, is evinced by the experience of this grower, whose 80,000 trees are all on trifoliate rootstocks. Two decades ago, after having had considerable trouble with exocortis, he adopted the practice of selecting budwood only from bearing trees on trifoliate rootstocks that showed no scaling or stunting. The trees that have come out of this program still show no symptoms of exocortis.

The failure to observe exocortis earlier in Florida may be attributed to the fact that trifoliate orange is rarely used in the main producing areas as a rootstock for oranges, tangerines, or grapefruit. All previous reports on exocortis have come from regions where trifoliata is employed for these varieties. In Florida this rootstock is almost wholly restricted to the growing of Satsumas in the northern counties. Exocortis has not been seen in this combination despite the fact that extensive observations have been made by workers familiar with this disease. This suggests, among other possibilities, that Satsumas have escaped infection, or that they are inimical to the presence or development of the causal virus.

The choice of citrus rootstocks that are tolerant to tristeza and that at the same time can be grown in wet, heavy soils, is critically limited. Trifoliata, possessing both these desirable qualities, has in the past received enthusiastic adoption by growers in tristeza-devastated areas. The subsequent experiences of these growers with losses from exocortis suggests that in Florida trifoliate orange should not be resorted to except on a limited or experimental basis until more is known about the occurrence of exocortis, both in its latent and expressed forms, in the citrus varieties of the State.

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STATE PLANT BOARD OF FLORIDA, CITRUS EXPERIMENT STATION, LAKE ALFRED, FLORIDA

OBSERVATIONS ON WHEAT MOSAIC IN VIRGINIA

C. W. Roane, T. M. Starling, and H. H. McKinney[1]

Soil-borne wheat mosaic appeared in Virginia in scattered fields of Atlas wheat in 1952 (1). It was reported from Virginia in 1925 when it occurred on Arlington Farm, near the site of the Pentagon Building (2), and it continued to appear there until 1941, when the farm was abolished. In 1952, it was detected on one farm each in Amherst, Buckingham, Essex, Halifax, Henrico, Prince Edward, and Westmoreland Counties and on several farms in Richmond County. In 1953, it was observed on single farms in Charlotte, Chesterfield, Hanover, and Smyth Counties and on several more farms in Essex and Richmond Counties. The total acreage known to be infested with mosaic does not exceed 500 acres, three-fourths of which is in Richmond County. The report from Smyth County is interesting in that the mosaic occurred in the same spot in a field from which meadow nematode (Pratylenchus) damage was reported in 1949 (5). The field was sown in 1949 to the Vahart variety which reacts mildly to mosaic. At that time, the reporter (Roane) had never seen wheat mosaic and the mild expression of mosaic led him to attribute the damage to meadow nematodes, which were present in the samples examined. In 1953, the field was sown to Atlas and the symptoms of mosaic were clear-cut. In general, the widespread appearance and recognition of wheat mosaic in Virginia may be coupled with the introduction and general acceptance of Atlas 50 and Atlas 66 wheats. Both are highly susceptible to soil-borne wheat mosaic. It is apparent now, especially following the Smyth County observations, that the virus causing wheat mosaic has existed for some time in Virginia soils, but general use of resistant varieties such as Forward, Leap, Leapland, Thorne, Vahart, and V. P. I. 131 has prevented its detection. In view of the fact that the growing of mosaic-susceptible wheats might result in considerable reduction in yield, it was deemed desirable to observe the performance of certain wheat varieties on virus-infested soil. For this purpose, a plot of virus-infested land for experimental use in Westmoreland County was generously supplied by the owner, W. D. Edwards.

Objectives

The objectives of the seedings were to determine the type of mosaic present as evidenced by symptoms on a series of tester varieties, and to determine the effect of mosaic on yield of several wheats that either are recommended for farm use or are being considered for recommendation. Additional seedings were made to observe the reaction to mosaic of certain breeding materials.

Procedure

The land selected for these observations was sown to Atlas 50 wheat in the fall of 1951. Mosaic developed in the spring of 1952 over about 75 percent of the area of the field. In some parts of the affected area plants were only mildly diseased but in others mosaic and stunting were severe. Samples taken from outside of the mosaic area yielded approximately 49 bushels per acre while those from an area in which symptoms were severe yielded approximately 13.5 bushels per acre. A plot 60 feet by 70 feet in a severely diseased area was chosen for future testing. Yield tests were sown in single 20-foot rows which were trimmed to 16 feet at harvest. Observations recorded included response to mosaic, percentage of plants showing symptoms, and yield in bushels per acre.

Two sets of data are presented. The observations from a group of tester varieties and some selections that have been resistant to soil-borne mosaic in Illinois and Indiana (3) are presented in Table 1. The observations on the varieties entered in the official Virginia yield tests are presented in Table 2. Average yields from the entries on virus-infested soil are compared with the yields from the same varieties on nearby virus-free soil. The results are not entirely comparative because the fertility level on the virus-free soil was higher and the yields are based on six replicates of three-row plots, whereas those from the virus-infested

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Table 1. Mosaic reactions and yield of

Table 1. Mosaic reactions and yield of certain tester varieties and resistant wheat selections on mosaic-infested soil, Westmoreland County, Virginia, 1953.

C.I. no.	Variety or selection	Replicate I			Replicate II			Average yield bu./A.
		Prevalence %[a]	Response[b]	Yield bu./A.	Prevalence %[a]	Response[b]	Yield bu./A.	
	*Harvest Queen	100	S(Ros.)[c]	0	100	S(Ros.)[c]	3.08	1.54
	*Harvest Queen sel. 34-3	95	M, Mo, S	11.00	90	M, Mo, S	9.24	10.12
	*Illinois 36-686 (Gladden x Minturki) x Gladden	T	M	31.24	T	M	31.68	31.46
11770	*Michigan Amber	100	VS	8.80	100	VS	8.36	8.58
	*Red Winter Spelt	100	VS	10.12	100	VS	10.12	10.12
	*Supresa x Redhart Strain 5	10	M, Mo	56.44	20	M, Mo	61.60	59.02
12220	*Indiana sel. 11512 (Trumbull x Fultz)	75	M, Mo	20.68	75	M, Mo, S	19.80	20.24
12557	Indiana Purdue 7 x Trumbull-Hope-Hussar-Fultz (sel. A339A2-2-2-6)	98	M, Mo, S	25.08	98	M, Mo, S	21.56	23.32
12798	Indiana Chinese-Michigan Amber	98	Mo, S	24.64	100	M, Mo, S	18.92	21.78
12534	Atlas 50 (Check)	100	VS	19.80	100	VS	10.56	15.18
12799	Indiana Kaw-W38-Fultz-Hungarian-Wabash-Fairfield-Trumbull	50	M, Mo, S	30.36	90	M, Mo	30.80	30.58
	Wabash x American Banner	98	Mo, S	17.60	98	M, Mo, S	15.84	16.72
13082	W38-Fultz-Hungarian-Wabash x Fairfield-Trumbull-Hope-Hussar	90	M, Mo	27.28	90	M, Mo	19.80	23.54
	Illinois 43-113C	95	M, Mo, S	21.56	95	M, Mo, S	22.88	22.22
12674	Saline, Illinois 1952 crop	90	M, Mo, S	26.40	95	M, Mo, S	28.16	27.28
	Y 165-48 (Y2267-46) BG 1-Trumbull	50	M, Mo, S	8.89	75	M, Mo, S	14.08	11.49

* The first seven entries have been used as test varieties in a number of fields in previous years by McKinney.

[a] Prevalence is an estimate of the percent of the plants showing mosaic symptoms.

[b] Response is graded into the following six categories: VS--very severe; S--severe; Mo-- moderate; M--mild; VM -- very mild; O —no mosaic.

[c] Ros.--rosette, characteristic response of Harvest Queen to wheat mosaic virus 1.

Table 2. Observations on adapted wheat varieties entered in the official Virginia yield tests on mosaic-virus-infested soil and yield on mosaic-virus-free soil, Westmoreland County, Virginia, 1953.

C.I. No.	Variety or selection[a]	Mosaic reaction[b]		Virus-infested soil			Virus-free soil		
		Prevalence	Response	Av. yield bu./A.[c]	Rank	Bu. test wt.	Av. yield bu./A.[d]	Rank	Bu. test wt.
6962	Nittany	100	S, VS	13.8	16	48.1	43.1	3	54.0
11856	Thorne	65	S	16.6	13	48.9	39.1	12	55.2
12185	Leap's Prolific (Leap)	100	M, Mo	18.5	11	50.3	32.6	20	51.7
12461	Trumbull x Frondoso (Y 2375)	97.5	Mo	36.4	2	53.9	40.5	11	54.7
12529	Seneca	55	Mo, S	16.0	14	50.2	41.8	6	55.5
12536	Atlas 50	100	VS	10.2	20	48.6	40.8	9	51.4
12536	Anderson	100	S	23.9	6	53.1	40.6	10	53.3
12537	Vahart	97.5	Mo, VS	11.6	18	49.2	44.2	1	55.3
12561	Atlas 66	100	VS	10.5	19	[e]	38.7	13-14	50.2
12651	Trumbull-W38-Fultz-Hungarian 128 x Trumbull3-Hope-Hussar	90	M, Mo	28.6	3	51.6	41.6	7	52.7
12658	Frondoso x Trumbull-Hope-Hussar (Ky. 37)	100	VS	13.2	15	48.2	42.6	5	52.3
12659	Frondoso x Trumbull-Hope-Hussar (Ky. 38)	100	VS	12.7	17	49.2	44.0	2	55.3
12755	Pennoll	100	Mo, S	19.2	9	51.0	38.0	15	55.4
12761	Leapland x Fronteira (Y 2381)	100	S	20.8	8	49.6	37.8	16	52.8
12798	Trumbull-Fultz sel. x Minhardi-Wabash-Purplestraw-Chinese-Michigan Amber (Indiana 414A29-4-8)	100	Mo	17.6	12	47.5	38.7	13-14	55.0
12985	W38-Fultz sel.-Hungarian-Wabash x Fairfield (Indiana 3917A5-11-2-1-1)	97.5	S	25.7	5	53.8	33.0	19	54.4
	Nudel	20	M, 0	47.0	1	54.7	36.9	17	55.1
	Vahart sel. 1	100	S	22.8	7	53.0	42.8	4	54.0
	Vahart sel. 10	100	S	19.0	10	52.0	41.1	8	54.5
	V. P. I. 131	57.5	M, Mo	26.4	4	53.9	36.5	18	55.0

[a] Atlas 50 was planted in every tenth row to check the distribution of the virus in the soil. It reacted uniformly with a prevalence of 100 percent and a response of VS.

[b] Response is graded into the following 6 categories: VS- very severe; S - severe; Mo - moderate; M - mild; VM - very mild; 0-no mosaic.

[c] Average of 3 replicates.

[d] Average of 6 replicates.

[e] No data but about equal to Atlas 50.

area are based on three replicates of single-row plots. However, they suffice to show the effect of mosaic on the yield of the varieties tested. On virus-infested soil, Atlas 50 (C.I. 125 34) was sown in every tenth row in order to determine whether the virus was uniformly distributed through the plots.

Results

Observations indicate that the mosaic-rosette virus (Marmor tritici var. typicum) (wheat mosaic virus 1) is present in the test soil since Harvest Queen rosetted and produced only a few spikes (Table 1). All other entries had mosaic symptoms without rosette. The extremely high degree of infection and severity of mosiac reaction that was obtained in this test nursery exceeded that previously observed in wheat mosaic variety test nurseries made over the years in Illinois, Indiana, and the Carolinas by one of the authors (McKinney). The six wheats and the spelt marked with asterisks in Table 1, that have been used as testers in many fields over a considerable period, all expressed a higher degree of mosaic than previously observed. Indiana Sel. 11512 from Trumbull x Fultz (C.I. 12220), that had failed to show mosaic in several field tests, finally did show signs of mosaic in McKinney's tests at Beltsville during the four seasons from 1950 to 1953, but the expression was evident in fewer plants and was less severe than in the Westmoreland County tests. In the case of Illinois Sel. 36-686, a single mosaic plant was observed by McKinney in a two-rod-row test at Clemson, South Carolina, in 1945, but it was strongly suspected that this was a volunteer rogue. Mosaic had not been observed in this wheat in the field prior to the test in Westmoreland County. Apparently this variety has the highest type of field resistance against the soil-borne wheat mosaic viruses that we have yet encountered. However, when inoculated by manual methods of juice transfer, it developed mosaic in a high percentage of the plants (3). Several other wheats that showed high resistance and tolerance to mosaic in tests in Illinois, Indiana, and South Carolina, developed an unusual amount of mosaic in the Westmoreland tests. However, in comparison with wheats having a record for high susceptibility over the years, the aforesaid wheats essentially retained their relatively high position for resistance.

No explanations are offered at this time for the exceptionally severe expression of mosaic in the Westmoreland County plots. If an increasing number of such fields appear in the future, it may be advisable to use Illinois Sel. 36-686 and similar wheats in breeding for mosaic resistance. However, for mosaic control in fields of the usual sort, most wheats that expressed mosaic of the order shown in Harvest Queen 34-3, Saline, and comparable lines or varieties in the Westmoreland plots, should be reasonably safe. Other wheats appear to be even safer (entries 3, 6, 10 in Table 1; entries 8, 9, 13, 19, in Table 2). In the test of varieties adapted to Virginia conditions (Table 2) there was a good correlation between resistance to mosaic and yielding ability on virus-infested soil. The virus apparently was distributed uniformly throughout the plot as was indicated by the yield and mosaic reaction of the check variety, Atlas 50. Except for Nudel, all entries yielded less on the virus-infested plots than on virus-free plots. The exceptionally high yields of Nudel and Supresa x Redhart Strain 5 may have resulted from reduced competition from surrounding varieties which were more susceptible to mosaic. All entries suffered a reduction in quality as is indicated by lighter bushel test weights from the virus-infested plots. On the basis of this test, Nudel, Y 2375, C.I. 12651, V.P.I. 131, and C.I. 12985, respectively, were highest yielding varieties in virus-infested soils but they were outyielded by other varieties in the absence of the virus. Additional yield tests will be needed to confirm these preliminary observations and to find the best varieties for mosaic-virus-infested soil in Virginia.

Significance

Soil-borne mosaic is a factor to be reckoned with in the production of wheat in Virginia. Several adapted varieties are available for production on mosaic-virus-infested soils. In the future it is important that varieties released to farmers should be highly resistant to mosaic. Also, resistance to mosaic should be a major factor in the breeding of new varieties of wheat for Virginia.

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COOPERATIVE INVESTIGATIONS BETWEEN THE VIRGINIA AGRICULTURAL EXPERIMENT STATION AND THE DIVISION OF CEREAL CROPS, UNITED STATES DEPARTMENT OF AGRICULTURE.

REACTION OF WHEAT VARIETIES AND SELECTIONS TO THE SOIL-BORNE VIRUSES IN SOUTHEASTERN UNITED STATES

J. G. Moseman, H. H. McKinney, and C. W. Roane[1]

Interest in the soil-borne viruses, mosaic-rosette virus, Marmor tritici var. typicum, and the yellow mosaic virus, M. tritici var. fulvum, has increased in Southeastern United States. The release of the varieties Atlas 50, Atlas 66, and Coker 47-27, which are very susceptible to mosaic, has helped to emphasize the need for resistant varieties. When those varieties were released, there was an increased interest in wheat as a cash crop and in improvements in cultural practices on farms.

These improved cultural practices included increases in the amount of nitrogen fertilizer applied. It then became apparent in fields where older susceptible varieties had done poorly that the soil-borne viruses and not the lack of nitrogen or other physical conditions were limiting production. At certain stages of growth it is difficult to distinguish between plants deficient in nitrogen and those infected with the yellow mosaic virus. Many varieties show no visible effects from the viruses unless the infestation is extremely high. Others, including the more recently released varieties, show symptoms of mosaic in proportion to the level of the virus in the field.

This report summarizes the more recent information on the reaction of wheat varieties and selections to the soil-borne viruses in Southeastern United States. Most of the entries either are varieties recommended by State Experiment Stations in the southeastern region or are

Table 1. Reaction of entries in the southeastern or southern uniform wheat nursery to the soil-borne viruses at Statesville, North Carolina in 1949, and 1952 and at China Grove, North Carolina in 1953.

C. I. No.	Variety or selection	Reaction[a] 1949 % Type	1952 % Type	1953 % Type
12534	Atlas 50	100 S	80 S	100 S
12561	Atlas 66	100 S	80 S	90 S
12461	Taylor	80 -	30 MO	10 S
12565	Hardired 47-12	100 VS	-- -	-- -
12333	Chancellor	0	tr.	40 S
12566	M925 (Maretts)	100 VS	-- -	-- -
1915	Purplestraw	100 S	80 MO	60 S
12026	Sanford	tr.	-- -	-- -
12294	Purcam	100 S	-- -	-- -
12563	Coker 47-27	-- -	80 S	95 S
12536	Anderson	100 MM	65 MO	20 S
12185	Leap	10 VM	tr.	0
6962	Nittany	100 MS	-- -	-- -
12537	Vahart	tr. VM	5 VM	-- -
12761	Leapland-Fronteira Y2381	-- -	60 MO	60 S
12463	Coastal	-- -	70 S	100 S
12658	Frondoso x THH Ky. 37	-- -	80 S	80 S
12659	Frondoso x THH Ky. 35	-- -	60 MO	30 S

[a] The degree of infection is indicated by the percentage of plants infected. The severity of the reaction on the plants is as follows: VS=very severe; S=severe; MO=moderate; MM=moderate to mild; M=mild; VM=very mild.

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Table 2. Reaction of entries in the uniform wheat mildew nursery to the soil-borne viruses at Statesville, North Carolina in 1952 and at China Grove, North Carolina and Westmoreland County, Virginia in 1953.

C. I. No.	Variety or Selection	Reaction[a]		
		N. C.		Va.
		1952 % Type	1953 % Type	1953 % Type
12184	Carala	80 S	100 S	100 S
2227	Chul	-- -	100 S	100 VS
8195	Axminister	0	0	15 MO
12664	Picardie	0	0	10 M
12665	Asosan	-- -	0	100 MO
11703	Sturgeon	-- -	tr.	100 MO
3315	Huron	-- -	5 S	100 MO
12411	Hardired 5	80 S	100 S	100 VS
12666	Suwon 92	tr.	tr.	90 M
12559	Trumbull[2]-Red Wonder-Steintim (Y3231)	20 M	tr.	100 MO
12670	Frondoso-Redhart[3]-Noll 28-Leap (Y2967)	80 S	100 S	100 VS
12671	Trumbull-Fronteira-Er. Ful.-Purplestraw (Y333)	tr.	0	5 M
11770	Michigan Amber Selection	80 S	100 S	100 VS
6902	Progress	-- -	10 S	100 MM
12747	Normandie	20 M	20 MO	65 MO
12991	Marfed-Merit, Sel. 2	40 M	tr.	85 S
12695	Canada 4232-20	-- -	100 S	100 VS
12633	Illinois 1-Chinese)[2] x Timopheevi-Wisconsin 245	-- -	90 S	100 VS
13086	Chancellor[2] x Timopheevi Hybrid Coker Sel. 788-126	-- -	0	100 VS
13087	Chancellor[2] x (Purcam-Chancellor-Suwon) Coker Sel. 781-2B	-- -	50 S	100 S
13088	Timopheevi Hybrid x Chancellor Coker Sel. 673-3B	-- -	60 S	100 S
12996	F. W. 806	tr.	tr.	50 M
12997	F. W. 815	0	0	10 M

[a] See Footnote a, Table 1.

being used in breeding as sources of resistance to powdery mildew (Erysiphe graminis), or leaf and stem rusts (Puccinia spp.). This article does not report the reactions of all varieties or selections tested by the authors, but does include most of the economically important varieties that have not been reported on previously[2].

Material and Methods

The seed used in these tests was supplied by various men in the Division of Cereal Crops and Diseases. H. A. Rodenhiser and the late C. V. Lowther furnished the seed from the Uniform Wheat Mildew and the Uniform Winter Wheat Rust Nurseries. The seed from the Uniform Southeastern or Southern Nursery and the International Rust Nursery were supplied by B. B. Bayles. The tests were conducted in cooperation with the Agricultural Experiment Stations in the State where the nursery was located[3]. In North Carolina tests were sown in virus-infested

[2] Koehler, B., et al. 1952. Soil-borne wheat mosaic. Univ. Illinois Bul. 556, 567-599.

[3] We wish to acknowledge the assistance given in these studies by T. T. Herbert and J. W. Hendricks in North Carolina and by T. M. Starling in Virginia.

Table 3. Reaction of entries in the uniform winter wheat rust nursery to the soil-borne viruses at Statesville, North Carolina in 1952 and at China Grove, North Carolina, and Westmoreland County, Virginia, in 1953.

C. I. No.	Variety or Selection	Reaction[a]		
		N. C.		Va.
		1952 % Type	1953 % Type	1953 % Type
5657	Trumbull	2 -	tr.	100 MO
3332	Mediterranean	2 -	tr.	100 MO
11384	Wabash	tr.	20 S	100 S
12992	Wabash x American Banner	tr.	0	60 S
12885	Indiana 39172A5-11-2-1-1	5 -	tr.	100 M
12993	Indiana 447A3-3-4	tr.	tr.	100 M
13083	Indiana B 40149A4-9-3-5	tr.	tr.	100 MO
12660	Warden-Leap	tr.	tr.	10 MM
4843	Hussar	80 S	100 S	100 S
11682	Hope-Hussar	30 MO	80 S	100 VS
5146	Kanred	50 MO	90 S	100 VS
1442	Karkof	60 MO	90 S	100 VS
4898	Malakof	50 MO	100 S	100 VS
12128	Ponca	30 MO	90 S	100 VS
12994	Kiowa x Marquillo-Oro	60 MO	80 S	100 VS
12800	Mediterranean-Hope x Pawnee[2]	60 MO	100 S	95 VS
12801	Comanche x Mediterranean-Hope x Chiefkan	60 MO	80 S	100 VS
12802	Bobin-Gaza-Bobin x Pawnee	70 S	70 S	100 VS
12803	T. Vulgare-T. Timopheevi x T. Vulgare[2]	70 S	100 VS	100 VS
12804	(Mediterranean-Hope-Pawnee) x (Oro Illinois 1-Comanche)	tr.	0	100 S
12145	Quanah	80 S	100 S	100 VS
12662	Minturki x T. Timopheevi	tr.	20 S	100 VS
12517	Comanche x Blackhull-Hard Federation	-- -	0	95 M
13090	Westar Sel.	-- -	10 S	100 S
12703	Sinvalocho-Wichita-Hope-Chev. x Wichita	-- -	100 S	100 VS
13091	Minturki x Timstein-Vulgare[2]	-- -	90 S	100 VS
13092	Yogo x (H44-Minturki, II-29-72)	-- -	95 S	100 VS
13093	(Illinois 1-Chinese x Timopheevi x Wisconsin Pedigree 2	-- -	10 S	100 VS

[a] See Footnote a, Table 1.

soils on the Piedmont Branch Experiment Station at Statesville from 1949 through 1952, and on the Roy Corriher farm near China Grove in 1953. The test in Virginia was near Warsaw in Westmoreland County on the farm of W. D. Edwards.

Only the maximum reading is reported where there were two replications or two readings. The degree of infection is indicated by the percentage of plants infected and the severity of the reaction of the plants by the type of reaction.

Results

As stated in the accompanying article[4], there was an extremely high degree of infection and severity of mosaic reaction of entries in the nursery in Westmoreland County, Virginia in 1953.

[4] Roane, C. W., T. M. Starling, and H. H. McKinney. 1954. Observations on wheat mosaic in Virginia. Plant Dis. Reptr. 38: 14-18.

Table 4. Reaction of 102 entries from the international wheat rust nursery to soil-borne viruses at China Grove, North Carolina, and Westmoreland County, Virginia, in 1953.

C. I. No.	Variety or Selection	Reaction[a]	
		N. C. % Type	Va. % Type
12053	Cadet	100 S	100 S
158406	Charter	0	100 M
131363	Dundee-RL 595 x H44-24-Marquis	tr.	0
12100	Egypt Na 101, PI 139599	100 S	100 VS
12894	Egypt Na 95, PI 153780	5 -	0
134044	Eureka	10 S	0
12470	Frontana	100 S	100 VS
12795	Gabo, PI 155431	0	80 M
8178	Hope	100 VS	100 VS
12633	(Illinois 1-Chinese)[2] Timopheevi, Wisconsin 245	80 S	100 VS
92472	Kenya Governor	100 S	100 VS
92473	Kenya Standard	0	0
124738	Kenya B. 286, C6042	5 S	100 M
12882	Kenya RF 324, C9906, PI 118896	tr.	0
118901	Kenya 130. B.8.F. CI0862	100 S	100 S
12568	Kenya 117A	100 VS	100 S
12880	Kenya 338.2C 2.E.2. PI 187165	0	95 M
177180	Kenya 338 AA.1.A.2	100 S	100 VS
177183	Kenya 341.0.2.B.1	80 S	95 S
-----	Mentana	100 S	100 S
177185	Kenya 350 AD.9.C.2	tr.	0
117789	Kenya-Florence-Dundee, 5148	tr.	0
150604	Maria Escobar	100 S	100 S
11876	McMurachy	0	100 S
12008	Mida	10 M	100 M
12318	Newthatch	100 VS	100 VS
12345	Red Egyptian RL 2061	5 M	100 M
168687	Rio Negro	100 S	100 VS
81790-2	Sapporo haru Komugi ichigo	0	tr. VM
12474	Surpresa, PI 103833	0	0
10003	Thatcher	80 S	100 VS
12347	Timstein	5 S	100 M
159098	No. 466-4-M-M-M	95 S	100 VS
159106	No. 43	0	100 VS
12635	Exchange	95 S	100 VS
117795	Kenya-Gular 5963	tr.	0
167660	------	100 S	100 M
170913	------	0	95 M
170918	------	0	100 M
178046	------	60 S	100 M
178062	------	95 S	100 M
185839	Garnet	100 S	100 VS
12876	Frontana x RL 2265-Redman[2], RL 2520	95 S	100 VS
-----	RL 2265 x Redman[2], RL 2518.1	100 S	100 VS
-----	RL 2265 x Redman[3], RL 2624	100 S	100 VS
-----	RL 2265 x Redman[3], RL 2769	-- -	-- -
-----	Frontana-Thatcher, II-46-3	100 S	100 S
-----	Timstein-Kenya 58, II-44-30	tr.	0
-----	Frontana-Thatcher, II-46-13	95 S	100 S
-----	Frontana-Thatcher, II-46-53	95 S	100 S
-----	Timstein-Henry, II-44-65	100 S	100 VS

Table 4 (continued)

C. I. No.	Variety or Selection	Reaction[a] N.C. % Type	Va. % Type
-----	Lee-Frontana, N. No. 2357	95 S	100 S
-----	Henry-Cadet, N. No. 2374	90 S	100 VS
-----	Supremo 211	0	tr.
-----	Supremo 51 A	0	25 M
13085	Kentana 52	70 MO	100 M
-----	Lerma 52	40 M	100 M
-----	Kenya[2]-Candeal, CR-244-3C-1c-1c-2c	0	tr.
-----	Egypt-Timstein, 704-1y-5y-3c-2c	100 S	100 VS
-----	(Egypt-Timstein) Mayo, 2156-2c-1c-6c	80 S	100 VS
-----	Egypt Na 101-Timstein x Mayo, 2156-6c-4y-1c-1y-1c	95 S	100 S
-----	Egypt-Kenya, 702-3y-2y-1c-1y-1c	100 S	100 VS
-----	Yaqui 50	100 S	100 VS
-----	Kentana-Yaqui, 2584-2y-7c	50 S	100 S
-----	Kenya-General Urquiza, 1121-11c-1y-1c-1c-1c	tr.	0
-----	(Kenya-Marroqui[2]) Maria Escobar, 1442-4c-1c-3c-2t	tr.	0
-----	Surpresa-(Kenya-Gular) 3707-89	tr.	0
-----	Renacimiento-Kenya, 3708-22	0	0
7265-5	Beladi. PI 57662	80 S	100 VS
3322	Pentad	tr.	100 S
12066	Stewart	tr.	100 VS
7067-1	Tremez Molle. PI 56258-1	10 S	100 VS
7065	Tremez Preto. PI 56256-3	10 S	100 VS
7066-1	Tremez Rijo. PI 56257-1	tr.	100 S
94701	CI 12898	90 S	100 VS
145720	------	80 VS	100 VS
109593	No. 214-30	60 S	100 VS
109597	No. 164-10	95 S	100 VS
191054	Caravaca 1	100 VS	100 VS
191076	Claro fino de Balazote	80 S	100 VS
191365	ST 464	80 S	100 VS
191395	T. persicum fuliginosum	10 M	0
192100	Addis Aleni	100 S	100 VS
192179	Amarai bianco tipo 142	90 S	100 VS
192502	Egypto 2100	80 MO	100 VS
-----	Barrigon (Yaqui)	90 S	100 VS
-----	Ld 194-Khapli x Ld 308, Ld 351	tr.	100 VS
-----	Carleton[2] x Ld 320, Ld 352	0	100 M
-----	Ld 316 x Nugget, Ld 353	70 S	100 VS
-----	Ld 246 x Ld 320-Carleton, Ld 354	tr.	100 M
-----	Chapingo	50 S	100 VS
-----	Ld. 308-Nugget, Ld 356	30 S	100 VS
-----	Ld. 308-Nugget, Ld 357	0	100 VS
174699	Glutinoso (durum)	tr.	100 S
12924-1c	Golden Ball-Iumillo-Mindum	100 VS	100 VS
12924-2c	Golden Ball-Iumillo-Mindum	100 VS	100 VS
12924-3c	Golden Ball-Iumillo-Mindum	100 VS	100 VS
3686	Vernal	60 S	100 VS
1526	Yaroslav	100 VS	100 VS
4013	Khapli	70 S	100 VS
94761-1	Timopheevi	100 VS	100 VS
94760	Timopheevi	100 VS	100 VS

[a] See Footnote a, Table 1.

Several entries that had been resistant in all previous tests were susceptible to the viruses present in this nursery. The readings on the material in 1953 in the North Carolina nursery were somewhat higher than in previous years. This may have been due to the new location of the nursery in 1953 or to the weather conditions during that season.

Most of the more recent entries in the former Southeastern and now Southern Nursery included in Table 1 are very susceptible to the soil-borne viruses. The Chancellor variety, which was highly resistant in 1949 and 1952, had 40 percent of the plants showing a severe type reaction in 1953. The Taylor and Anderson varieties had a lower percentage of plants infected in 1953, but the type of reaction was more severe. These changes in reaction indicate there may have been a different strain or strains of virus present at Statesville than at China Grove. It may also be the result of different growing conditions. Leap and Vahart were resistant in all three tests.

The reaction of the entries from the Uniform Wheat Mildew Nursery, when grown in the North Carolina mosaic nurseries in 1952 and 1953 and in Virginia in 1953, is given in Table 2. The varieties Picardie, Suwon 92, C. I. 12671, Progress, Normandie, F. W. 806, and F. W. 815 showed condiderable resistance in all the nurseries in which they were tested. Axminister, Asosan, Sturgeon, Huron, and C. I. 12559 were resistant to the virus strains in the North Carolina nurseries and showed some tolerance in the Virginia nursery. The other varieties in the mildew nursery were very susceptible in at least one of the tests.

The reaction of the entries from the Uniform Winter Wheat Rust Nursery when grown in the mosaic nurseries in North Carolina in 1952 and 1953 and in Virginia in 1953 is given in Table 3. Several Indiana selections, which had been resistant in previous tests, showed a high degree of infection and a moderate type reaction in the Virginia nursery. The Warden-Leap selection, C. I. 12660, was the most resistant variety in this nursery. Trumbull, Mediterrean, Wabash-American Banner, C. I. 12885, C. I. 12993, C. I. 13083, C. I. 12804, and C. I. 12517 gave resistant reactions in the North Carolina nurseries but had a high degree of infection and moderate to severe type reaction in the Virginia nursery. The other varieties and selections were susceptible in practically all the nurseries in which they were tested.

The 102 entries listed in Table 4 are from the International Rust Nursery. The type of reaction for most of the entries in that nursery was more severe in the Virginia nursery. Some entries were resistant at both locations. Some of the varieties such as Egypt Na 95, Gabo, Kenya Standard, Kenya 338.2C 2.E.2., and Supremo 51 A, that are being used in breeding at the present time, were found to be highly resistant. Other breeding lines such as Frontana, Hope, Mentana, Exchange, and Surpresa were susceptible. When a plant breeder uses some of the latter lines in breeding, it would be advisable to test the progeny for their reaction to mosaic.

Summary and Conclusion

This report includes the reactions of wheat varieties and selections to the soil-borne viruses in Southeastern United States that have been observed over a period of years in North Carolina and Virginia. Included are most of the varieties now being grown in the Southeastern States. Also the best known sources of resistance to powdery mildew, and leaf and stem rust were included. Several of these have been used in breeding programs where the two soil-borne viruses, mosaic-rosette virus and yellow mosaic virus, are known to be present.

If more fields with a high level of infectivity like that near Warsaw, Virginia are found, it may be necessary to use other sources of resistance to this disease. At the present time, it appears that those entries that were resistant in the North Carolina Nurseries should be adequate for most fields.

COOPERATIVE INVESTIGATIONS BETWEEN THE DIVISION OF CEREAL CROPS AND DISEASES, BUREAU PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING, U. S. DEPARTMENT OF AGRICULTURE, AND THE NORTH CAROLINA AND VIRGINIA AGRICULTURAL EXPERIMENT STATIONS.

GERMINATION TESTS WITH COMMERCIAL LOTS OF WHEAT SEED TREATED WITH COPPER CARBONATE

Alvin Overland and W. L. Nelson[1]

Winter wheat seeded in Lincoln County, Washington in the fall of 1952 germinated and emerged during the month of January, 1953. The temperature during the time of germination and emergence was about 5° C. The stands in many fields were poor and irregular and it was noted that most of these fields were from seed which had been treated with copper carbonate by the slurry method. This suggested that copper carbonate applied in slurry form has a deleterious effect on the viability of wheat seed. To check this point samples of seed were obtained from two local seed processing plants, one of which had used the dust treatment and the other the slurry treatment. Samples of untreated seed were obtained to be used as a check. These samples of treated and untreated seed were from lots of seed distributed to farmers. The seed had been treated at the recommended rate of two ounces per bushel.

Materials and Methods

Two germination tests were made at different times. These were carried out in the Seed Laboratory at the State College of Washington. An attempt was made to simulate field conditions by subjecting part of the seed to a cold treatment. This treatment consisted of placing the seeds on moist blotters in petri dishes in a refrigerator held at 3° C. for a period of 19 days in the first test and 11 days in the second test. During these periods the petri dishes containing the seeds were removed from the refrigerator to room temperature for an average of several hours each day. The period of exposure to room temperature was about an hour per day at first and then was increased to four or five hours toward the end of the exposure periods. The wheat was then transferred to a germinator maintained at a temperature of 20° C. This temperature is standard for germinating wheat.

A comparable germination test was made with seed not subjected to the cold treatment. Preliminary sprout counts were made after five days and final counts were made after ten days. Only normal healthy sprouts were recorded.

Percentage of germination was calculated on the basis of 100 seeds planted, each planting being replicated two and four times respectively, in the first and second test. The results are presented in Table 1.

Table 1. Results of germination tests with winter and spring wheat seed treated with dry and slurry forms of copper carbonate showing a differential effect on viability of the seed germinated at 20° C.

Variety[b]	Percent[a] germination at indicated fungicide and temperature treatments.					
	Check		Dust		Slurry	
	3° C. Exposure[c]	None	3° C. Exposure	None	3° C. Exposure	None
Elgin	98*	95	91	95	73	77
Elmar	93*	92	89	93	53*	77
Turkey	95	95	90*	96	75	83
Hymar	87	90	96*	93	37*	79
Federation	98	93	96	94	85	83
Idaed	90	93	-[d]	--	87	86

[a] Average of two separate tests, except that asterisk denotes figures for one test only.

[b] The last two listed are spring wheats.

[c] The seed was incubated at 3° C. for 11 to 19 days prior to incubation for germination at 20° C.

[d] No test.

[1] Seed Analyst and Assistant Agronomist, respectively, Washington Agricultural Experiment Station, Pullman, Washington.

As shown in Table 1, wheat treated with slurry copper carbonate gave lower percentages of germination than untreated seed or seed treated with copper carbonate dust. In the first test the reduced germination of copper carbonate slurry-treated samples was more marked at 3° C. than at 20° C. However, at both temperatures, germination of wheat treated with copper carbonate slurry was markedly less than either untreated seed or seed treated with dry copper carbonate. The second test produced the same general differential among the three treatments but the germination percentages were higher.

With respect to varieties, the winter wheats were more adversely affected by the copper carbonate slurry treatment than the spring wheats. This was especially true at 3° C.

These results suggest the need for more thorough testing of fungicides before making recommendations on different methods of application. It has been assumed that copper carbonate slurry would, like the dust treatment, be non-toxic to wheat seed. According to these data copper carbonate slurry is injurious to wheat seed, particularly under low temperature conditions.

WASHINGTON AGRICULTURAL EXPERIMENT STATION, PULLMAN

STEMPHYLIUM LEAF SPOT OF ALFALFA IN ONTARIO[1]

W. G. Benedict[2]

Heretofore, in Canada, the foliage disease of alfalfa (Medicago sativa L.) caused by the fungus Stemphylium botryosum Wallr. (Pleospora herbarum (Pers. ex Fr.) Rabenh.) had been reported only in British Columbia. In 1940 (1), Stemphylium leaf spot was reported to be more prevalent than the common leaf spot caused by Pseudopeziza medicaginis (Lib.) Sacc. in the locality of Agassiz, and in 1943 (2), it was fairly general on alfalfa in the lower mainland of British Columbia, although damage was slight.

In early September, 1953, an examination of a stand of three-year-old alfalfa in test plots at the Science Service Laboratory, Harrow, Ontario, disclosed the occurrence of Stemphylium botryosum[3] and considerable damage was indicated. The leaf lesions were numerous, brown in colour with a lighter center, irregular in shape, 1-2 mm. wide, and each was surrounded by a straw-coloured halo (Fig. 1). Under field conditions appreciable defoliation occurred, as shown by the development of the disease on an alfalfa plant illustrated in Figure 2. The seriousness of the damage to the foliage at that time of year was apparent as the autumn growth of the crop had just begun.

The causal fungus, which is characterized by echinulate muriform conidia (Figs. 3, 4), and a perithecial stage, was identical with Stemphylium botryosum as described by O. F. Smith (3). Conidiophores and conidia were produced abundantly on leaf lesions.

The fungus was readily isolated in pure culture from field material by placing small pieces of diseased leaf tissue on acidified potato-dextrose agar in poured Petri dishes. Cultures were white at first but, after a few days, developed greenish black pigment. Immature perithecia appeared abundantly in the cultures in about 7 to 12 days, but did not develop asci even after two months.

The pathogenicity of the organism was proven by inoculating alfalfa plants in the greenhouse and reisolating the fungus from the leaf lesions that were produced. A heavy suspension of conidia and mycelium of the fungus was prepared by placing a 3 x 1 1/2 cm. piece of culture in 35 c.c. of distilled water in a Waring blender operated for five minutes. After a sticker-spreader was added, the suspension was applied to leaves by means of an atomizer and eye-dropper. Plants were placed in a cheesecloth-covered chamber held at a high atmospheric humidity in the greenhouse for 72 hours before being moved to a greenhouse bench.

Daily microscopic examinations of several inoculated leaves showed that after 36 to 48 hours numerous spores had germinated and the hyphae had advanced 50 to 75 micra over the leaf surface. After 72 hours a considerable amount of new mycelium was seen on the leaves and pale areas appeared where the fungus apparently had infected the leaf tissue. Brown spots containing conidia and conidiophores were produced five to seven days after inoculation. Two days later, under the greenhouse conditions prevailing in early October, the affected leaves dropped. Infection occurred more frequently on new growth of seedling and first-year alfalfa plants than on that of two- or three-year-old plants. The fungus also proved to be pathogenic on young red clover plants. Elongate brown lesions producing the dark brown echinulate conidia borne on the typically nodulose conidiophores (as shown in Fig. 3) appeared on a few leaves three to five days after inoculation. Young tomato plants, similarly inoculated, were not infected.

An intensive survey of Essex and nearby counties on September 10 showed that Stemphylium leaf spot of alfalfa was quite prevalent locally and occurred sparsely in Huron County. Damage caused by this foliage disease seemed greatest in fields of alfalfa growing on sandy soils when the crop was coming into bloom after a dry summer season. This leaf spot contributed appreciably to the defoliation in many alfalfa fields that were severely damaged by the black stem disease caused by Ascochyta imperfecta Pk.

The Stemphylium disease of alfalfa probably has escaped notice in Ontario prior to this year. Its prevalence this year seemed to be associated with a prolonged period of hot, humid weather. Spread of the disease from older to younger leaves (Fig. 2) in the field was especially evident in flowering and seeding crops. These had undergone a natural weakening during a summer drought. Artificial infection readily occurred in new foliage of alfalfa of various ages transplanted from the field to the greenhouse.

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[1] Contribution No. 1333, from the Botany and Plant Pathology Division, Science Service, Department of Agriculture, Ottawa, Canada.

[2] Associate Plant Pathologist.

[3] Identification of the fungus was confirmed by Dr. J. W. Groves, Principal Mycologist, Ottawa.

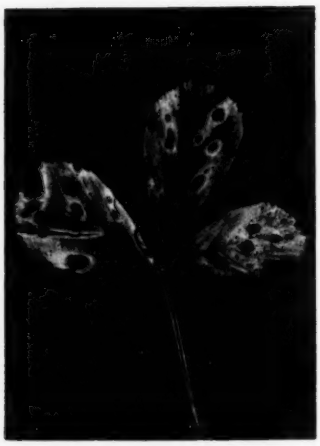

FIGURE 1.

FIGURE 2.

FIGURE 3.

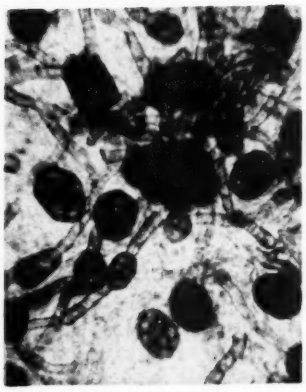

FIGURE 4.

FIGURE 1. Leaf lesions on alfalfa caused by Stemphylium botryosum. x 1 1/2.

FIGURE 2. Alfalfa plant showing intensive natural infection by Stemphylium leaf spot. x 3/8.

FIGURE 3. Typical nodulose conidiophores and attached muriform conidium (x about 675) of Stemphylium botryosum on naturally infected alfalfa leaf.

FIGURE 4. Dark, echinulate, muriform conidia (x about 300) of Stemphylium botryosum on artificial medium.

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DISEASES OF FORAGE CROPS IN NEW YORK, 1953

Daniel A. Roberts, Karl D. Fezer, and Robert T. Sherwood

Meadows and pastures in 23 New York counties were inspected in June and August, and losses resulting from diseases were estimated after infection percentages and severity ratings had been obtained (5, 7). In most cases, fields were visited by one or more persons, but estimates of losses in 12 fields were made after inspection of representative samples of plants which had been collected by co-operators[1].

Spring blackstem of alfalfa and northern anthracnose of red clover were the most destructive stem and foliage diseases of forage crops observed in 1953 (Table 1). A period of warm, dry weather preceding the first cutting of hay was sufficient to check the spread of blackstem, and serious losses in alfalfa fields were averted. The epiphytotic of northern anthracnose brought about complete loss of some mammoth red clover seed crops, and caused substantial reductions of seed and forage yields in most medium red clover fields.

Other stem and foliage diseases of legumes and grasses resulted in crop losses either less than or about the same as those reported previously (5, 7). Although bacterial wilt is a serious disease in some alfalfa fields, it is adequately controlled in most areas by the use of resistant varieties. Losses due to virus and complex root and crown diseases could not be determined accurately. However, these diseases certainly appear to be important factors in stand maintenance problems throughout New York State, and probably are among the chief causes of losses in clover fields.

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Footnotes for Table 1. (on opposite page).
[a] Number of fields inspected before first cuttings (left), and prior to second cutting (right).
[b] Common names are those used by Dickson (1) except where names are followed by parenthetical numbers which refer to literature cited.
[c] Numbers based on total number of fields inspected.
[d] Loss not determined.
[e] Apparently caused by Fusarium spp.

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[1] Thanks are due to Eric J. Kresse, A. A. Johnson, and Keith Norton for collecting specimens.

Table 1. Estimated crop losses due to some diseases of forage legumes and grasses in New York, 1953.

CROP: No. fields inspected[a] Disease[b]	First cutting		Second cutting	
	No. fields showing disease	Estimated loss[c] %	No. fields showing disease	Estimated loss[c] %
ALFALFA: 16, 14				
Black stem (Ascochyta imperfecta)	16	3.5	7	1.0
Common leafspot (Pseudopeziza medicaginis)	14	2.0	13	2.0
Stagonospora leafspot (Stagonospora sp.)	3	0.1	6	0.4
Stemphylium leafspot (Pleospora herbarum)	3	trace	3	trace
Yellow leaf blotch (Pyrenopeziza medicaginis)	0	---	1	trace
Downy mildew (Peronospora trifoliorum)	1	trace	2	trace
Leaf rust (Uromyces striatus var. medicaginis)	0	---	2	trace
Mosaic virus diseases	4	---[d]	6	---[d]
Crown bud rot (4) (Complex of soil-borne fungi)	2	---[d]	2	---[d]
Bacterial wilt (Corynebacterium insidiosum)	2	---[d]	4	---[d]
RED CLOVER: 9, 14				
Northern anthracnose (Kabatiella caulivora)	7	4.0	12	8.0
Pseudopeziza leafspot (5) (Pseudopeziza trifolii)	7	2.5	7	1.5
Stemphylium leafspot (Stemphylium sarcinaeforme)	2	0.2	7	0.5
Sooty blotch (Cymadothea trifolii)	0	---	3	trace
Leaf rust (Uromyces trifolii var. fallens)	2	trace	9	0.6
Mosaic virus diseases	4	---[d]	6	---[d]
Root and crown rots (6) (various)	9	---[d]	14	---[d]
WHITE AND LADINO CLOVERS: 17, 4				
Pseudoplea leafspot (Pseudoplea trifolii)	4	0.1	4	0.2
Cercospora leafspot (5) (Cercospora zebrina)	3	trace	3	trace
Mosaic virus diseases	13	---[d]	2	---[d]
Crown and stolon rots[e]	14	---[d]	1	---[d]
BIRDSFOOT TREFOIL (LOTUS SPP.,): 11, 8				
Stemphylium leafspot (3) (S. loti)	11	0.6	8	0.7
Sclerotinia crown rot (S. trifoliorum)	1	trace	0	---
Crown and root rots[e]	5	---[d]	0	---
TIMOTHY: 7, 3				
Heterosporium leafspot (5) (H. phlei)	7	1.0	3	1.0
Leaf streak (Scolecotrichum graminis)	6	0.4	1	0.2
Helminthosporium leaf blight	3	trace	1	trace
Leaf rust (Puccinia poae-sudeticae)	1	trace	2	trace
ORCHARD GRASS: 6, 2				
Leaf streak (Scolecotrichum graminis)	5	0.4	2	0.6
Leaf scald (Rhynchosporium orthosporum)	4	1.0	2	0.6
Leaf rust (Puccinia rubigo-vera)	0	---	2	trace
Purple leafspot (2) (Stagonospora maculata)	6	0.8	2	0.4
BROME GRASS: 9, 2				
Helminthosporium leaf blight (Pyrenophora bromi)	9	1.5	2	1.0
Leaf scald (Rhynchosporium secalis)	4	1.0	1	trace

See footnotes for Table 1. on opposite page.

EFFECT OF FUNGICIDES AND SPRAYING SCHEDULES UPON INTERNAL MOLD OF CHILE[1]

Philip J. Leyendecker, Jr.[2]

New Mexico produces a considerable amount of sun-dried chile[3], which demands a premium price because of its high pungency and bright color. Kiln-dried chile is very dark red and does not impart the bright color, which, in Southwestern cookery, is characteristic of the sun-dried product. Many shipments of New Mexico sun-dried chile, upon reaching market, have been found to contain large numbers of pods which contain internal molds. Most of these moldy pods cannot be detected by external examination. Figure 1 shows three moldy pods that have been split open to expose a few fungi which were responsible for internal mold. Twenty-one different species of fungi have been isolated from moldy pods in New Mexico (1). From 80 to 90 percent of the damage was caused by different species of Alternaria, Fusarium, and Hormodendrum. These fungi develop mainly after the first killing frost, when 40 to 60 percent of the crop is still fleshy and subject to injury. It has been shown that after such frosts the pod wall, at the calyx end, pulls away from the seed mass (2). This creates a space for air-borne spores to enter the injured pods, and the pod cavity provides excellent conditions for the germination and establishment of the air-borne fungi.

FIGURE 1.
Split pods of Capsicum annuum L., showing three of the most common fungi responsible for internal contamination in New Mexico:
A. Fusarium roseum.
B. Hormodendrum cladosporioides.
C. Alternaria tenuis.

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[2] Associate Plant Pathologist, New Mexico Agricultural Experiment Station, State College, New Mexico.

[3] Also commercially known as red pepper, chile is the dried fruit of Capsicum annuum L. Chili is another spelling, especially in trade circles.

Internal mold is a serious problem confronting the chile industry in New Mexico because shipments of moldy pods have been refused or down-graded, giving the entire production of sun-dried chile from the State an unfavorable reputation.

A plausible control measure appeared to be the application of certain commerical fungicides to fruited plants in the field. The New Mexico Agricultural Experiment Station undertook such a three-year study, the results of which follow.

Materials and Methods

Small, randomized-replicated field plots were established on the New Mexico A & M College horticultural farm. All plots were field seeded to a local strain of chile known as College No. 6, a large-fruited, thick-walled selection which is very susceptible to internal mold. The following fungicides, to which a spreader-sticker was added, were applied with a small pressure sprayer at the strengths indicated: Phygon XL 1 lb. per 100 gal. water (US Rubber); Zerlate 1 1/2 lbs. per 100 gals. (DuPont); Dithane 1 1/2 lbs. per 100 gal. (Rohm & Haas); Copper-Hydro 6 lbs. per 100 gals. (Chipman); and dehydroacetic acid (DHA-S) 1% (Dow). The following spraying schedules were followed: A, One application October 15. B, Two applications, October 15,and October 30. C, One application; day after killing frost. D, Two applications, day after killing frost and one week later.

After spraying, all pods remained on the plants to sun-dry. When the pods were field cured, usually about December 15, they were harvested and examined in the laboratory by splitting open each pod and scoring for the presence or absence of fungi.

Results and Discussion

Table 1. Effect of field application of fungicides for control of internal mold of chile, Capsicum annuum.

Fungicide	Percent better than check: Year 1950	1951	1952	Average	Rank
1. Phygon XL	22	15	17	18	1
2. Dithane	15	10	13	13	3
3. Zerlate	14	12	13	13	2
4. Copper-Hydro	12	7	16	12	4
5. Dehydroacetic acid	5	0	7	6	5

Table 2. Effect of time of application upon control of internal mold of chile, Capsicum annuum.

Treatment	Percent better than check: Year 1950	1951	1952	Average	Rank
A. One application before frost	10	11	9	10	4
B. Two applications before frost	14	10	11	12	2
C. One application day after frost	17	15	20	17	1
D. Two applications day after frost and one week later	8	14	12	11	3

Tables 1 and 2 show that internal mold of chile can be appreciably controlled by applying fungicides to fruited plants in the field. Of the materials tested, Phygon XL gave the best results, followed by Zerlate, Dithane, and Copper-Hydro. The latter three fungicides gave almost equal control. Dehydroacetic acid was not effective in controlling internal mold. Internal mold cannot be entirely eliminated by the field application of fungicides. However, the fungicides decreased appreciably the extent of internal and external fungal contamination of the pods. The mold colonies in treated pods were invariably restricted in size, and the external mold growth on the pedicel and calyx was materially reduced. This in turn would make for a mold-free ground chile, since the calyx and pedicel are usually ground in the preparation of chile powder.

The timing of the fungicide applications appeared to influence the efficiency of control (Table 2). The best control was obtained when the succulent pods were sprayed the day following the first killing frost. An additional spray application one week later did not seem to afford additional protection from mold contamination. Sprays applied before frost were probably not as effective because they reduced the spore inoculum potential for a period following spraying, but afforded little protection at frost time, when pod injury occurred.

Summary

1. Internal mold, a marketing problem of sun-dried chile produced in New Mexico, is caused primarily by species of Alternaria, Fusarium, and Hormodendrum.
2. The percentage of moldy pods was appreciably reduced by spray application of fungicides to fruited plants in the field.
3. Phygon XL gave the best control, followed by Zerlate, Dithane, and Copper-Hydro, with little or no control from dehydroacetic acid.
4. Of the four spraying schedules tested, one application applied the day after frost gave the best results.

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TOMATO GREY MOLD IN PAPALOAPAN, VERACRUZ, MEXICO[1]

William D. Yerkes, Jr.[2], Ralph W. Richardson, Jr.[2],
and Oscar Brauer Herrera[3]

In February and March of 1953 an attack of tomato grey mold disease (Cladosporium fulvum Cke.) on field-grown tomatoes occurred in Papaloapan, Veracruz, Mexico. The field contained lines of tomatoes being studied genetically. The tomatoes had been transplanted on November 20, 1952, and the disease was first noted on February 21, 1953. There was a continuous spread of the disease in the field until March 23, 1953, at which time the experiments were terminated.

Table 1. Reaction of tomato varieties and selections to grey mold attack in a field planting, Papaloapan, Veracruz, Mexico.

Variety of origin	Type of growth	Susceptibility
Rep. II-PL. 4 Northrup King	Determinate	Very susceptible
Rep. II-PL. 10 Selections		Very susceptible
Rep. II-PL. 8	Determinate	Very susceptible
Maui H.E.S. 3380	Determinate	Very susceptible
Hawaii	Determinate	Very susceptible
Southland	Indeterminate	Moderately susceptible
Desc. 23	Indeterminate	Moderately susceptible
San Marzano	Indeterminate	Moderately susceptible
Stokescross #2	Indeterminate	Moderately susceptible
Hoja de Papa	Indeterminate	Moderately susceptible
Long Red	Indeterminate	Resistant
Red Jacket	Indeterminate	Resistant
Stokescross #2 (F_2 #1)	Indeterminate	Resistant

The weather in the month of February was mild and cloudy. Twenty days were recorded as overcast or partly cloudy. Fifteen mornings were recorded as foggy, and appreciable dewfall was noted on 21 days. Rain fell on 9 days, with the heaviest precipitation on February 13, when 9.8 mm were recorded. The maximum temperature for the month was 26.4°C, and the minimum 17.5°; the average daily temperature was about 22°.

March weather was warmer and drier. In the first 15 days of March, 5 days were cloudy, rain fell in trace amounts on 2 days, there were no foggy days, and only 4 days were recorded as having appreciable dewfall. The highest temperature observed was 28.2°C, the lowest 23.5°, with the average falling around 26°. These weather conditions are close to the optima determined for the fungus by other workers.

A list of the varieties and the severity of attack is given in Table 1. It is to be noted that plants with indeterminate type of growth were less susceptible. This may be due in part to the more open type of plant, allowing for more rapid drying of the lower leaves.

The disease appeared most markedly on the plants bearing a heavy set of fruit. Also, the earliest varieties were the most severely attacked. Disease appeared gradually in later varieties as they bore more fruit, usually in conjunction with the approximation of heaviest production.

MEXICAN AGRICULTURAL PROGRAM OF THE ROCKEFELLER FOUNDATION, AND OFFICE OF SPECIAL STUDIES OF THE MEXICAN MINISTRY OF AGRICULTURE AND ANIMAL INDUSTRY

[1] Journal Series Paper No. 9 of the Mexican Agricultural Program of The Rockefeller Foundation.
[2] Staff members of the Mexican Agricultural Program of The Rockefeller Foundation.
[3] Office of Special Studies, Mexican Ministry of Agriculture and Animal Industry.

OCCURRENCE OF VERTICILLIUM WILT IN ARIZONA

Ross M. Allen[1]

A survey has been made during the past summer to determine the occurrence of Verticillium wilt (Verticillium albo-atrum) in each of the cotton-producing counties of Arizona. The disease is considerably more prevalent in the State than had been realized. Approximately 5.7 percent (37,000 acres) of the cotton land planted to short-staple varieties is wilt-infested with a resulting reduction of the total yield by about 2 percent. Substantial infestations, not previously reported, are located in six counties: Cochise, Graham, Greenlee, Pima, Pinal, and Santa Cruz. Small infestations have been observed in Maricopa County, while an inspection of 85 fields in several widely separated areas of Yuma County failed to locate this malady, possibly because higher soil temperatures prevalent in that area are unfavorable for the pathogen.

Two distinct types of wilt have been observed. On land in cultivation for the first year severe wilt in cotton may occur as discrete, roughly-circular spots varying from two to ten yards in diameter. Plants in the center of the circle are usually severely stunted, defoliated, and die early in the growing season. From the center towards the edge of the circle the cotton plants appear progressively less affected. Field history and personal observations have shown that spots of this type practically disappear in the second year of cropping to cotton since only an occasional infected plant may be found; in the third year the spots, in several cases, have disappeared completely. The discrete-spot type of occurrence somewhat parallels the "fluctuating" type occurrence reported in hops by Isaac and Keyworth (1).

In contrast to the transitory or fluctuating incidence of wilt is the more general infection that may occur over entire fields but is usually most prevalent at the lower end of the field where irrigation water frequently accumulates. Infestations of this more or less permanent type correspond to the "progressive" type of wilt of hops, also described by Isaac and Keyworth, in that the incidence of the disease is increasing with each year that cotton is grown in the infested soil. Typical foliar symptoms of Verticillium wilt are observed though severe defoliation and killing of plants seldom occur until near the end of the growing season.

Soil type has not been correlated with the incidence of the discrete-spot type of wilt which seems to occur in any of the Arizona cotton soils, but a small degree of success has been obtained in predicting the occurrence of the permanent type of wilt. Soils of the Pima, Gila, and Cajon series, having friable subsoils without definite accumulation of lime, especially the silted phases, are found associated with the latter type occurrence.

An unusual complex of Verticillium wilt and Texas root rot (Phymatotrichum omnivorum), which substantially reduced the yield of the affected acreage, was observed near Eloy in Pinal County. The two diseases were of such general occurrence in one 80-acre field of short-staple cotton that it was difficult to determine which disease had actually killed individual plants since practically every plant in the field had been attacked by one and in many cases by both of the pathogens. Dual incidence of these diseases makes prescription of control measures quite difficult because most of the commonly acceptable controls for either of the diseases will make conditions more favorable for and encourage increase of the other.

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JUTE STEM CANKER IN CUBA[1]

Bowen S. Crandall[2]

Among the fiber crops being tested in Cuba, there are a considerable number of jute selections. Both the round-pod (white) variety, Corchorus capsularis L., and the long-pod, C. olitorius L., have been brought in at various times from Egypt, Syria, Turkey, India, and Pakistan. Until July 1952 there was no record of any disease affecting the selections of jute under trial.

Shortly after the beginning of the wet season in 1952, when the plants were a month old, a severe stem-canker disease developed in the plantings. This disease is characterized, in the fields, by a wilting of large groups of seedlings. This wilting is found to be caused by black girdling stem cankers of various lengths, though most of them are at least 2 to 3 inches long. (Figure 1).

FIGURE 1. Jute stem canker caused by Sclerotium bataticola in Cuba. A. Appearance of disease in field. B. Detail of the stem canker.

Tissue isolations from the cankered areas yielded 100 percent of the fungus Sclerotium bataticola Taub. Although the isolates from jute differ somewhat from isolates of this species from a root disease found in the same area on kenaf, Hibiscus cannabinus L., taxonomically they are the same species. No opportunity has yet presented itself to compare cultures, but it seems possible that this stem-canker disease is the same as that reported from India as being caused by Macrophomina phaseoli (Maubl.) Ashby, the pycnidial stage of S. bataticola.

Under local conditions five strains of C. capsularis (PI-150186, 187292, 187293, 187295, and 190452) were highly susceptible to the stem-canker disease, and one (PI-190453) was resistant. All 13 selections of C. olitorius under trial (PI-180025, 180035, 180046, 181239, 181240, 181241, 181242, 181746, 181745, 181869, 183219, 183253, and 187295) are apparently

[1] A contribution from the Cooperative Fiber Commission, Havana, a technical agricultural organization operated jointly by the Governments of Cuba and the United States. United States participation in this work was carried out as part of the technical cooperation program in Cuba, administered by the Foreign Operations Administration.

[2] Pathologist, Foreign Agricultural Service, U. S. Department of Agriculture, assigned to the cooperative work in Cuba.

resistant. Individual plant inoculations of these selections will be carried out to select canker-free lines.

Early in June 1953 the season's first hurricane resulted in unusually heavy rainfall in the experimental fields. Losses were experienced in the rotations of velvet beans, Mucuna deeringiana (Bort.) Small. The damage was due to black girdling cankers, extending several inches above and below the soil line; from these cankers strains of S. bataticola similar to those from jute were isolated.

COOPERATIVE FIBER COMMISSION, HAVANA, CUBA

NEMATODES ASSOCIATED WITH MINTS IN OREGON[1]

C. E. Horner and H. J. Jensen

Previous accounts of nematodes associated with the genus Mentha are few and widely scattered in the literature. In 1938, Buhrer (1) listed Mentha piperita and M. arvensis var. piperascens as hosts for Meloidogyne. Goodey (2) listed Aphelenchoides ritzema-bosi on M. piperita and Aphelenchoides olesistus on M. spicata in his 1940 host catalog. In 1951, Henderson (3) stated that Ditylenchus destructor was found infesting the roots of M. arvensis by A. D. Baker in Canada. In Michigan and Indiana, Dieter[2] found Meloidogyne hapla and Paratylenchus spp. associated with M. piperita.

During 1952 and 1953 several nematodes have been found closely associated with or causing disease in mints in Western Oregon. Observations on Meloidogyne hapla, Paratylenchus spp., Aphelenchoides spp. and Longidorus sp. are reported here.

A root-knot nematode (Meloidogyne hapla) was first found on Scotch spearmint (Mentha cardiaca) which had been transplanted from the field to greenhouse pots. Subsequent examination of the field planting revealed that Scotch spearmint was infected with root-knot nematodes while in adjacent fields no infection was apparent on peppermint.

In greenhouse inoculation tests, root knot-infested soil was used to expose several mint species and varieties to infection. The test plants were three species of commerically grown mints, two native "wild" mints, and 15 hybrids of Mentha piperita X M. crispa[3]. Cuttings from

Table 1. Mint species, varieties, and hybrids infected with Meloidogyne hapla in greenhouse tests.

Species, variety, or hybrid	Number of plants	
	Inoculated	Infected
Common spearmint (Mentha spicata)	5	5
Scotch spearmint (M. cardiaca)	10	10
Commercial peppermint (M. piperita var. Mitcham)	10	10
"Wild" peppermint (M. piperita var. americana)	5	5
Field mint (M. arvensis var. canadensis)	5	5
Pennyroyal (M. pulegium)	5	5
M. crispa	8	8
Hybrid No. 88-35	5	5
Hybrid No. 88-36	5	5
Hybrid No. 88-40	5	5
Hybrid No. 88-46	5	5
Hybrid No. 88-47	5	5
Hybrid No. 88-57	5	5
Hybrid No. 88-60	5	5
Hybrid No. 88-94	5	5
Hybrid No. 88-96	5	5
Hybrid No. 88-101	5	5
Hybrid No. 88-103	5	5
Hybrid No. 88-109	5	5
Hybrid No. 88-112	5	5
Hybrid No. 88-119	5	5
Hybrid No. 107	5	5

[1] Approved for publication as Technical Paper No. 817 by the Director of the Oregon Agricultural Experiment Station. Contribution of the Department of Botany & Plant Pathology.

[2] Personal communication from C. E. Dieter of the Dow Chemcial Company.

[3] The 15 hybrids are part of the many hybrids being developed for resistance to Verticillium wilt and mint rust by Dr. C. A. Thomas, Bureau of Plant Industry, Soils, and Agricultural Engineering.

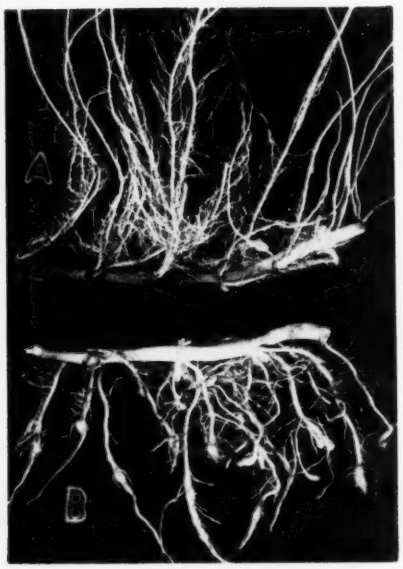

FIGURE 1. Comparison of healthy (A) and root knot-infected (B) roots of Scotch spearmint. (Photograph by H. H. Millsap)

FIGURE 2. Paratylenchus macrophallus female partly dislodged from surrounding peppermint leaf tissue. (Photograph by H. K. Phinney)

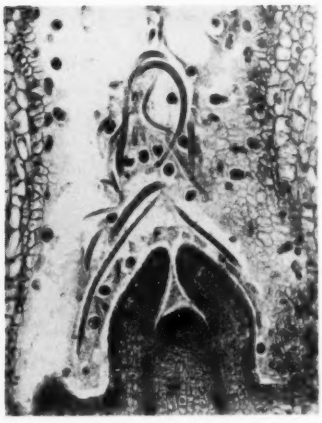

FIGURE 3. Apehlenchoides sp. in a longitudinal section of a peppermint stem tip.

FIGURE 4. Root die-back condition of peppermint caused by Longidorus sp. (Photograph by H. H. Millsap)

healthy plants were rooted in clean sand and transferred to clay pots of infested soil. Control plants were grown in steamed soil. Results are given in Table 1.

All species and varieties of mints tested were infected by the root-knot nematode. Larvae and gravid females were found in all mints tested 68 days after being exposed to infection. Control plants were not infected with nematodes. Figure 1 shows a comparison of healthy and root knot-infected roots of Scotch spearmint.

Paratylenchus macrophallus has been observed in all stages of development within living stems and underground buds of commercial peppermint from most fields examined in Western Oregon. Fungi causing a previously reported root and rhizome rot (4) are closely associated with large numbers of Paratylenchus in the mint tissue and surrounding soil.

Soil around stunted and chlorotic plants in one field contained 8,000 Paratylenchus per quart and the nematodes were observed in the root tissue. Figure 2 shows a Paratylenchus macrophallus female partly dislodged from the surrounding living leaf tissue.

Aphelenchoides parietinus and other species of Aphelenchoides have been found associated with the meristematic cells of underground mint shoots. As many as 400 specimens of Aphelenchoides were counted in a single shoot tip. The nematodes occupy the spaces between the apical meristem and leaf primordia, and under the young leaves enveloping the apical meristem. Several days after shoots have emerged from the soil, the nematodes are no longer found in the tips. Figure 3 shows a longitudinal section of the growing tip of a mint shoot (M. piperita) with numerous nematodes.

During 1953 a species of Longidorus was found to be associated with extensive stunting and dying of peppermint throughout many of the older mint growing areas of Western Oregon. In experimental tests Longidorus caused stunting and a root die-back condition on peppermint. Figure 4 shows the root die-back condition and may be compared with the healthy roots in Figure 1.

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OREGON AGRICULTURAL EXPERIMENT STATION, CORVALLIS

CLITOCYBE ROOT ROT FOUND WIDESPREAD AND DESTRUCTIVE IN GEORGIA AND SOUTH CAROLINA PEACH ORCHARDS

Arthur S. Rhoads

Georgia and South Carolina are the only States in the Southeast from which information concerning the occurrence of Clitocybe root rot was lacking prior to the latter part of 1952. Hesler and Whetzel[1] mentioned Georgia as one of several States from which this disease had been reported on apple trees. This information apparently was based on old Plant Disease Survey records which the writer had been unable to verify more than ten years ago. Certainly Hesler and Whetzel's inclusion of California and Oregon in its distribution, and their statement that this is a disease of the Southwest, were both in error.

The writer has been interested in Clitocybe root rot for 30 years. In the summer of 1941 he made a special effort to find it in southwestern Georgia, but without success. During October, 1952, in the yard of a residential property at Athens, he found fresh clusters of sporophores of Clitocybe tabescens on the root of a large water oak (Quercus nigra), which had been felled shortly before because of decay by Polyporus dryadeus.

On October 7, near Union, South Carolina, the writer observed desiccated sporophores of C. tabescens at the base of the stump of a planted hardwood tree of unknown species. On the following day, in company with Dr. W. A. Campbell of the Division of Forest Pathology, a peach orchard at Cross Anchor, Spartanburg County, South Carolina, was noticed in which several trees were declining or dead on the margin of an area from which a considerable number of trees had been removed. A brief examination revealed that the declining trees were attacked by C. tabescens, and two desiccated sporophore clusters were found at the base of one tree. The fungus was isolated from a piece of root cut from one of the attacked trees on which it had not fruited.

A small cluster of sporophores that had developed at the base of a peach tree after it had been dug up in the Station orchard and moist-chambered in the greenhouse, brought to Dr. Julian H. Miller by Dr. E. F. Savage, Horticulturist of the Georgia Agricultural Experiment Station at Experiment, Georgia, was identified as C. tabescens by the writer. Dr. Savage stated that the disease caused by this fungus apparently occurred widely throughout the peach belt of Georgia since trees that seemed to be affected had been found at Fort Valley, Marshallville, and Zebulon. The disease had been observed for a considerable time at both Experiment and Fort Valley, but the cause of the trouble was not recognized until the fall of 1952. Previously it had been attributed either to winter injury or to damage following the use of chemicals for borer control. Digging around affected trees to a depth of 6 inches below the ground level at Experiment failed to reveal any injury to the root crown and it was not until the soil was removed to depths ranging from 8 to 14 inches that the mycelial sheets of the fungus were found under the bark. Clusters of sporophores of C. tabescens developed at the bases of five of these trees in this orchard in September, 1952. In another peach orchard at Experiment C. tabescens attacked three-to-four-year-old trees that had made an exceptionally vigorous growth, including one tree each of Elberta peach on Yunnan and Shalil rootsocks, both of which are favorably regarded because of their resistance to root-knot nematodes (Meloidogyne).

In order to get some idea of the distribution and prevalence of Clitocybe root rot in Georgia, a number of peach orchards in the central part of the State were visited through the cooperation of Dr. Savage, as well as of Dr. John H. Weinberger, Senior Horticulturist of the U. S. Department of Agriculture Horticultural Field Crops Laboratory at Fort Valley, the largest peach-growing area in the State. Clitocybe root rot was found on from one to a few trees in each of six orchards inspected. In some orchards the disease had killed replants as early as the second year. In one orchard, in digging around a replant, pieces of peach roots infected by C. tabescens were found that had been left in the ground when the previous tree was removed. The only evidence of C. tabescens fruiting on the attacked trees examined consisted of rotting sporophore clusters found at the base of one. The characteristic appearance of the whitish to cream-colored mycelial sheets of this fungus between the bark and the wood, as it occurred in one of these replants, is illustrated in Figure 1. The orchards examined, with the exception of one on a sandy soil, were on clay or sandy clay. Clitocybe root rot was widespread throughout a large part of an orchard on a stony clay soil on a hillside three miles east of Woodbury, Merriwether County. Trees in this orchard had been reported dying several years previously and it had been abandoned three years ago. Most of the dead and dehorned trees showed abundant

[1] Hesler, L. R., and H. H. Whetzel. 1920. Manual of fruit diseases, p. 102.

FIGURE 1. Peach replant girdled by Clitocybe tabescens during the second year's growth in an orchard at Fort Valley, Georgia. Bark cut away to show the characteristic mycelial sheets of the fungus in the cambial region. About 9/20 (almost half) natural size.

evidence of Clitocybe root rot, though several had been attacked so long that they had become invaded by various wood-rotting fungi and the bases and roots had deteriorated too far to be sure that Clitocybe was the cause of death. The characteristic mycelial sheets and strands of C. tabescens had developed abundantly between the bark and the wood of the root crown and roots on 22 trees that were dug around in a part of this orchard, and sporophores, nearly all old and desiccated, were found at the bases of 14 of these trees. Three additional trees still living likewise showed Clitocybe root rot. In another abandoned orchard higher up the hillside on the same property Clitocybe root rot was found on three dead trees and on one still partly living, with old sporophore clusters of C. tabescens occurring at the bases of two of the dead trees.

An orchard seven miles east of Griffin, Spalding County, in which trees set out to replace dead trees had proved short-lived, afforded an unusual opportunity to examine a considerable number of dead or declining trees that had just been pulled. A total of 18 of these trees, of which 16 were dead or nearly so, had been attacked by Clitocybe root rot. The disease was found also on two unpulled trees with some dead limbs. In no case, however, were sporophores of the fungus found. In one part of another large 17-year-old orchard, five miles east of Griffin, three trees with dead limbs were attacked. It was difficult to locate instances of root rot in this orchard because of gumming and dying of the bark at the bases of many trees following injury by the peach borer, and decay by various wood-rotting fungi in the bases of others.

In an orchard at about 850 feet elevation at Rex, Clayton County, the original trees were well over 30 years of age but many had been removed and replanted over the years. Two trees out of several that had been removed about a year before were found to show old mycelial sheets of C. tabescens in the roots.

A small section of an orchard five miles east of Jonesboro, also in Clayton County, in which six-year-old trees had been planted in an extension onto newly cleared forest land where several oak stumps had been left, was also visited. Clitocybe root rot was found on two declining trees and had girdled two others recently pulled. The fungus was fruiting at the base of one of the living trees. Several trees in the vicinity of those showing root rot had been removed and replanted that year. This was the only peach orchard on newly cleared land visited. All other orchards examined for root rot were planted on land that had been cleared many years ago, some having been in peaches continuously for from 17 to 35 years.

A few orchards were examined in the northeastern section of Georgia. At the Horticultural Farm of the University of Georgia at Athens loss of peach trees had occurred over the years from an unidentified cause. Inspection of the scattered trees remaining in a 20-year-old block revealed that the root crowns and lateral roots of two of the three living trees selected for digging around were extensively invaded by C. tabescens. In a nearby eight-year-old orchard the fungus was attacking two living trees and five of the holes from which several trees had been removed earlier in the fall, contained infected peach roots, thus showing that these trees had succumbed to root rot. The method of tree removal practiced here was to cut the roots in a small circle and then dig a hole of like size. When new trees are planted at places where trees have died from root rot without taking the precaution of removing the old diseased roots there is little need to speculate as to why the replants should prove short-lived. Such lack of orchard sanita-

tion can only result in a gradual build-up of the sources of inoculum in the older orchards and create conditions increasingly favorable for infection.

In a 14-year-old orchard at Watkinsville, Oconee County, the root crowns and roots of four living trees and two that had died during the year were extensively attacked by C. tabescens. The affected trees occurred at widely scattered points in the orchard, but mostly adjoining local areas where one or more trees had died and been removed previously.

In a ten-year-old orchard at Farmington, farther south in Oconee County, Clitocybe root rot was found on five dead or dying trees at various points.

Locating instances of root rot by the general appearance of the trees was rendered difficult by the lateness of the season and the partial defoliation that had occurred already in some orchards. Since winter injury occurred in many orchards visited in central Georgia; trees showing this injury were avoided. The difficulty of locating attacked trees was greatly increased also by the unusual depth to which trees were planted in some cases and by the fact that the depth of the soil above the root crown was increased by working towards the trees in cultivation, and mounding for borer control. The prolonged general drought of the summer and fall was not conducive to the development of sporophores of the root-rot fungus, though fairly good rainfall occurred in Spalding County during the latter part of October.

From this preliminary survey in central and northeastern Georgia, it is evident that Clitocybe root rot is widespread, destructive, and obviously of long-standing in the State. Some of the results of this survey, for which the writer performed the pathological work, have already been presented.[2]

In the latter part of October the writer returned to South Carolina to make a preliminary survey of peach orchards in that State to determine whether or not the disease occurred in other orchards than the one at Cross Anchor, where it was found previously. Through the cooperation of Prof. A. M. Musser and Dr. H. H. Foster of the South Carolina Agricultural Experiment Station at Clemson, the writer was enabled to examine orchards in the principal peach-growing sections of that State. Mr. D. H. Petersen, Pathologist of the U. S. Department of Agriculture, located at Clemson, joined in the trip to the Piedmont section. Unfortunately, owing to a prolonged drought, the soils were extremely dry and, because of this, no freshly developed sporophores of C. tabescens were found. The general practice of growers to mound the soil around the bases of the trees for borer control, and frequently to plant trees deeply, made it necessary to dig the soil from around the bases of the trees examined, as in Georgia, in order to determine whether or not Clitocybe root rot occurred. Trees showing some evidence of decline, especially in areas where other trees had been removed, were selected for examination. A few well-advanced cases of Clitocybe root rot were found in each of three orchards visited at Inman, Spartanburg County, which has the greatest concentration of peach trees of any county in the State. These orchards were on clay soils in the Piedmont section. In one of these orchards the mycelium of C. tabescens was found in roots remaining in the ground from a tree that had been removed in an area where several trees had been pulled.

On the following day orchards were visited at various points in the Sandhill section of the State. Trees attacked by Clitocybe root rot were found in two orchards about six miles west of Lexington, and in another near Edmond, nine miles south of Lexington, all in Lexington County. In a portion of one of these orchards only five or six years old, where a large percentage of trees already had been removed and replanted, Clitocybe root rot was found on two dead trees and also on two replants that had died before the end of the first year in the orchard. Close to one of these replants, the writer found peach roots with an extensive development of the mycelial sheets of C. tabescens that had been left from the tree removed.

Clitocybe root rot was also found in two orchards in Saluda County, one near Monette and the other near Ridge Spring. In the latter examination of an area where the majority of the trees were dying or dead or had been removed led to finding Clitocybe root rot on eight dead original trees and also on two replants that had died shortly before. One of these had died at the end of the second year and the other early in the fourth year in the orchard. Old desiccated sporophore clusters of C. tabescens were found at the bases of two of the eight original trees. These cases of root rot were found scattered through a large area where the disease was unusually widespread and destructive.

In three orchards visited in Edgefield County only a single case of Clitocybe root rot was found. All these orchards apparently had been in peaches or some other crop for many years

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[2] Savage, E. F., J. H. Weinberger, E. S. Luttrell, and A. S. Rhoads. 1953. Clitocybe root rot, a disease of economic importance in Georgia peach orchards. Plant Dis. Reptr. 37: 269-270.

after the land was cleared.

This brief preliminary survey in South Carolina establishes that Clitocybe root rot is of widespread occurrence in the principal peach-growing sections in both the Piedmont and Sandhill regions and indicates that, here too, it has been an important factor in tree loss for years, and especially in the early decline of trees planted in orchards where trees attacked by the causal fungus were removed. Unfortunately, the limited time available did not permit determining the incidence of the disease in any one orchard. In view of its economic importance, more detailed surveys are highly desirable in both States. Such surveys doubtless will show a much wider distribution and frequency of occurrence of the disease than is here indicated.

The finding that Clitocybe root rot occurs more or less frequently in a number of orchards or parts of orchards examined at random at widely scattered points in both Georgia and South Carolina establishes a good reason why peach trees frequently prove short-lived, especially when planted where others have died. However, it is not suggested that, aside from virus diseases such as phony peach, this is the sole contributing cause for early mortality, though it unquestionably is a very important one.

In connection with the recent discovery that Clitocybe root rot is of widespread occurrence in the principal peach-growing areas of both Georgia and South Carolina, it is of interest to note, as pointed out by the writer[3], that this disease was known to be destructive to peach and other orchard trees in Oklahoma for several years prior to 1901, and in Missouri, Arkansas, and Florida as far back as 1902. Evidence that it is native to timbered lands has been found in a number of southern States, and this appears to be the case also in Georgia and South Carolina. In addition to the instances previously noted at Athens, Georgia, and Union, South Carolina, the writer found C. tabescens fruiting in October, 1952, on buried remains of some hardwood tree in a partly cleared tract of woods on the grounds of the Georgia Agricultural Experiment Station at Experiment, and at the base of a living boxelder (Acer negundo) in the woods along a branch of the Oconee River at White Hall, a few miles southeast of Athens. The closely related root-rot fungus Armillaria mellea was observed to develop frequently at the bases of forest trees in Georgia, particularly at Athens, but it was not observed on peach trees either in this State or South Carolina.

While Clitocybe root rot may be expected to occur in orchards planted on recently cleared land where hardwood trees occurred, the occurrence of this disease on land that has been cultivated and planted to peach trees for many years is of unusual interest. The writer's experience has shown that infection by C. tabescens usually takes place through contact of roots with old infected roots in which the fungus can develop saprophytically. It does not appear that the inception of this disease in orchards on land that has been cleared and cultivated for many years can be explained on this basis. However, so far as is known, there is nothing to indicate that this root-rot fungus can maintain itself and spread through the soil in the absence of old roots. Further studies are desirable to determine whether infection can take place in some other way, as appears to be the case.

Owing to the nature of Clitocybe root rot, it is much easier to combat this disease by taking preventative measures in advance rather than endeavoring to apply remedial ones after it has developed. In orchards where it is found to occur, every effort should be made to locate and remove all attacked trees rather than to allow them to remain until they become unproductive or die. This should prove very helpful in preventing the further spread of the disease to adjacent trees. With favorable soil moisture conditions, it should often be possible to locate attacked trees, particularly in the fall of the year, by the development of sporophore clusters of the causal fungus at the bases, provided the soil has not been mounded or worked up to an unusual depth. To minimize the possibilities of early infection of replants set out in places where root rot-affected trees are removed, it is highly important that growers take every precaution to remove all roots, preferably by the use of a grubbing-hoe, after the trees are pulled. It should be borne in mind that even small pieces of infected roots left in the ground can harbor the root-rot fungus and frequently serve as possible sources of infection for several years. With the adoption of this precaution and the elimination of diseased trees from the orchard, it should be reasonably safe to replant. While aeration and drying of such infected roots as are removed will soon kill the fungus, gathering and burning them will insure that they will not be worked into the ground again.

During the preliminary surveys in Georgia and South Carolina the writer isolated C. tabescens from the roots of attacked bearing trees and recently dead replants in orchards as

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[3] Rhoads, Arthur S. 1950. Clitocybe root rot of woody plants in the southeastern United States. U. S. Dept. Agric. Circ. 853.

follows:

Georgia: One at Experiment, Spalding County; three at Fort Valley, Peach County; and one each at Athens, Clarke County, and Watkinsville, Oconee County. He also examined two isolates of the fungus made by Dr. E. S. Luttrell, Plant Pathologist of the Georgia Agricultural Experiment Station, from roots of a peach tree and spores from a sporophore developed on a peach tree, respectively, at Experiment.

South Carolina: One at Cross Anchor and two at Inman, Spartanburg County; one near Lexington, Lexington County; and one near Ridge Spring, Saluda County.

Wood-rotting fungi found attacking the trunks and main limbs of peach trees in these States include Polyporus cinnabarinus, P. gilvus, P. hirsutus, P. submurinus, P. tulipiferus, P. versicolor, Stereum sp., and Ganoderma curtisii. The latter, which is often found growing from the roots or bases of trees, was observed on peach trees at Gainesville, Florida, in former years. It is not certain whether this is a primary or a secondary fungus on peach trees.

DIVISION OF MYCOLOGY AND DISEASE SURVEY, BUREAU OF PLANT INDUSTRY, SOILS AND AGRICULTURAL ENGINEERING.

HOSTS OF PHYTOPHTHORA CINNAMOMI RANDS

William A. Thorn and George A. Zentmyer

The soil pathogen Phytophthora cinnamomi was first described in 1922 when R. D. Rands reported its isolation from stripe cankers of cinnamon trees in Sumatra (42). In the intervening 30 years this fungus has been reported on a wide variety of hosts, in many different parts of the world.

The first definite record of P. cinnamomi in the United States is R. P. White's report of the fungus on rhododendron in New Jersey in 1930 (52). In 1932 a species of Phytophthora was reported as the causal agent of a root rot of chestnut and chinquapin trees in the southeastern United States (34); the fungus was later identified as P. cinnamomi (14). In 1935 P. cinnamomi was found to be the cause of a destructive root rot of red pine in nursery seedbeds in the southeastern United States (26). Crandall, Gravatt, and Ryan theorized that the dying of chestnut and chinquapin trees in this area, reported as early as 1824, may have been from the same root disease caused by P. cinnamomi that they described in 1945 (19). They further speculated that P. cinnamomi may have been introduced into the southern United States in the early nineteenth century, possibly by means of importation of exotic tropical or semitropical plants from Asia and the East Indies. Once establishing a foothold in the southern ports and on plantations the fungus may have spread slowly northward and westward by means of the susceptible native hosts, chestnut, chinquapin, and pine. Campbell recently has reported P. cinnamomi on shortleaf pine in the Appalachian region (12).

Another early report of this fungus as the causal agent of a root disease is the description by Tucker (47) of the fungus on avocado in Puerto Rico in 1929. In 1942 Wager (50) reported isolation of P. cinnamomi from avocado in California. Many additional reports have established this fungus as a serious and common pathogen on avocado in Florida, Texas, Mexico, Honduras, Costa Rica, Peru, South Africa, and Australia.

Other major hosts on which the fungus causes a severe root or crown rot include cinchona in Guatemala and Malaya, pineapple in Hawaii and Australia, heather in England and the United States, camellia in the southern United States and California, papaya in Peru, and many different types of coniferous and broad-leaved nursery stock in the southeastern United States.

Following is a list of the hosts of P. cinnamomi, obtained from a survey of the literature to July 1953:

SCIENTIFIC NAME	COMMON NAME	REPORTED FROM	REFERENCE
	Coniferous Trees		
*Abies nordmanniana	Fir, Nordmann	Southeast U.S.	18
*Abies alba (pectinata)	Fir, Silver	Southeast U.S.	18
*Abies sibirica	Fir, Siberian	Southeast U.S.	18
*Cedrus atlantica	Cedar, Atlas	Southeast U.S.	18
*Cedrus deodara	Deodar, cedar	So. Calif.	59
*Chamaecyparis lawsoniana	Cypress, Lawson	So. Calif., Oregon	46, 59
*Cupressus sempervirens	Cypress, Italian	So. Calif.	56
Cupressus sp.	Cypress	Argentina	23
*Juniperus sp.	Juniper	Maryland	27
*Larix decidua	Larch, European	Southeast U.S.	14, 19
*Larix leptolepis	Larch, Japanese	Southeast U.S.	14, 19
*Libocedrus decurrens	Incensecedar, Calif.	So. Calif.	56
*Picea abies (P. excelsa)	Spruce, Norway	Southeast U.S.	14
*Picea pungens	Spruce, Colorado	Southeast U.S.	14
*Pinus canariensis	Pine, Canary	So. Calif.	59
Pinus echinata	Pine, Shortleaf	Southeast U.S.	12, 13, 25, 35
Pinus radiata	Pine, Monterey	Australia, Argentina, So. Calif.	3, 23, 59
*Pinus resinosa	Pine, Red	Southeast U.S.	14, 19, 26
*Pinus sp.	Pine	Australia	7
*Pinus sylvestris	Pine, Scotch	Southeast U.S.	14

SCIENTIFIC NAME	COMMON NAME	REPORTED FROM	REFERENCE
	Coniferous Trees (cont.)		
Pinus taeda	Pine, Loblolly	Southeast U.S.	35
*Taxus baccata	Yew, English	Southeast U.S.	14, 19
*Taxus baccata (var. fastigiata)	Yew, Irish	Oregon	46
*Taxus cuspidata	Yew, Japanese	Southeast U.S.	14, 19
*Taxus media	Yew, Anglojap	Southeast U.S.	19
*Taxus sp.	Yew	Maryland	27
*Thuja occidentalis compacta	Arborvitae, Parsons	So. Calif.	56
Thuja sp.	Arborvitae,	Argentina	23
	Evergreen Broadleafed Trees		
Aleurites fordii	Tung oil tree	Southern U.S.	28
Carica papaya	Papaya	Peru	10
Cinchona officinalis (C. ledgeriana)	Quinine	Peru, Guatemala, Malaya	16, 20, 45
Cinchona pubescens (C. succirubra)	Quinine	Peru, Guatemala, Malaya	16, 20, 45
Cinnamomum burmanni	Cinnamon, Malay	Sumatra	42
Citrus aurantium	Orange, Sour	Brazil	22
Eucalyptus spp.	Eucalyptus	Australia	3
Lithocarpus densiflorus	Tanoak	Southeast U.S.	19
Ocotea architectorum	Ocotea	Peru	11
*Olea sp.	Olive	Australia	4
Persea schiedeana	Aguacate, Chinini	Mexico	58
Persea americana	Avocado	Puerto Rico, So. Calif., Peru, Costa Rica, Honduras, South Africa Mexico, Australia	7, 15, 17, 31, 47, 50, 55, 56, 57, 58
	Deciduous Broadleafed Trees		
*Betula alba	Birch	Southeast U.S.	14, 18
*Betula papyrifera	Birch, Paper	Southeast U.S.	14, 18, 19
**Castanea alabamensis	Chinkapin, Alabama	Southeast U.S.	19
**Castanea alnifolia	Chinkapin, Trailing	Southeast U.S.	19
**Castanea ashei	Chinkapin, Ashe	Southeast U.S.	19
***Castanea crenata (japonica)	Chestnut, Japanese	Southeast U.S.	14, 19
Castanea dentata	Chestnut, American	Southeast U.S.	14, 34, 48
**Castanea henryi	Chinkapin, Henry	Southeast U.S.	19
**Castanea margaretta var. arcuata	Chestnut	Southeast U.S.	19
*Castanea mollissima	Chestnut, Chinese	Southeast U.S.	14
*Castanea ozarkensis	Chinkapin, Ozark	Southeast U.S.	14
**Castanea pumila	Chinkapin, Allegheny	Southeast U.S.	19, 34
Castanea sativa	Chestnut, European	Southeast U.S., England	1, 21
Castanea sp.	Chestnut	Spain, Portugal,	40, 49
Fagus sp.	Beech	England	21, 38, 54
Jacaranda sp.	Jacaranda	Australia	3, 9
*Juglans nigra	Walnut, Eastern Black	Southeast U.S.	14, 19
*Juglans regia	Walnut, Persian	Southeast U.S.	14, 19
*Platanus orientalis	Plane, Oriental	Southeast U.S., Argentina	14, 23

SCIENTIFIC NAME	COMMON NAME	REPORTED FROM	REFERENCE
	Deciduous Broadleafed Trees (cont.)		
*Prunus armeniaca	Apricot	Australia	4
*Prunus persica	Peach	Australia	2, 4, 7, 9
*Prunus sp. (Mariana + Myrobolan stocks)	Plum	Australia	4
Punica sp.	Pomegranate	Australia	3
*Quercus alba	Oak, White	Southeast U.S.	14, 19
*Quercus borealis	Oak, Northern Red	Southeast U.S., France	14, 19, 43
*Quercus montana	Oak, Chestnut	Southeast U.S.	14, 19
*Quercus robur	Oak, English	Southeast U.S.	18
Quercus sp.	Oak	So. Calif.	36
Quercus suber	Oak, Cork	Southeast U.S., Portugal	18, 40
*Robinia pseudoacacia	Locust, Black	Southeast U.S.	14
	Shrubs, Perennials, Annuals		
Ananas comosus (sativus)	Pineapple	Hawaii, Australia, New South Wales, Queensland	6, 29, 30, 32, 33
**Antirrhinum majus	Snapdragon, common	England	38, 54
*Azalea mollis	Azalea	Australia	4, 5
*Azalea sp.	Azalea	Maryland, Australia	7, 9, 27
Baeckea brevifolia		Australia	3
*Camellia japonica	Camellia	Alabama, Florida, Louisiana, So. Calif.	24, 41, 51, 59
*Camellia magnoliaeflora	Camellia	Australia	3
*Camellia sasanqua	Camellia	Florida	51
*Camellia sp.	Camellia	Australia	7, 9
**Calceolaria sp.	Calceolaria	England	38, 54
*Chamaelaucium uncinatum		Australia	3, 4
Dillwynia ericifolia		Australia	3
Embothrium coccineum	Chilean Firebush	Australia	3
Epacris microphylla	Australiaheath	Australia	3
Epacris pulchella	Australiaheath	Australia	3
*Erica hiemalis	Heather	England	39
*Erica nivalis	Heather	England	39
Erica regerminans	Heather	California	44, 59
*Erica willmoreana	Heather	England	39
Eriostemon crowei	Eriostemon	Australia	3
*Ilex crenata	Holly Japanese	Maryland	27
Leucopogon microphyllus		Australia	3
*Lilium philippinense	Lily, Philippine	Australia	3, 4
**Mathiola sp.	Stock	England	54
Micrantheum ericoides	Micrantheum	Australia	3
*Myrtus (communis) compacta	Myrtle, Compact	So. Calif.	59
**Nicotiana glutinosa	Tobacco	England	38, 54
Phyllota phyliocoides		Australia	3
Pultenaea elliptica	Pultenaea	Australia	3
Rhododendron californicum	Rhododendron	New Jersey	53
***Rhododendron carolinianum	Rhododendron	New Jersey	53
**Rhododendron catawbiense	Rhododendron	New Jersey	53
**Rhododendron caucasicum (var. Boule de Niege)	Rhododendron	New Jersey	53
Rhododendron indicum			

SCIENTIFIC NAME	COMMON NAME	REPORTED FROM	REFERENCE
	Shrubs, Perennials, Annuals (Cont.)		
(Azalea indicum))	Rhododendron	Australia	8
**Rhododendron maximum	Rhododendron	New Jersey	53
*Rhododendron mucronulatum	Rhododendron	Southeast U.S.	19
*Rhododendron ponticum	Rhododendron	New Jersey	52, 53
Rhododendron sp.	Rhododendron	Australia, Argentina, Maryland	7, 23, 27
**Richardia scabra (Richardsonia scabra)	Mexican clover	Hawaii	33
**Ricinus communis	Castorbean	Hawaii	33
**Schizanthus sp.	Butterfly flower	England	38, 54
*Senecio sp.	Cineraria	England, Scotland	37
Sprengelia incarnata		Australia	3
Styphelia longiflora		Australia	3
*Viburnum sp	Viburnum	Maryland	27
**Vicia faba	Broad Bean	England	54

* Reported only on nursery stock.
** Reported infected by artificial inoculation.
Note: Standarized Plant Names, 2nd Edition, 1942, used as reference for scientific and common names.

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DEPARTMENT OF PLANT PATHOLOGY, UNIVERSITY OF CALIFORNIA, CITRUS EXPERIMENT STATION, RIVERSIDE, CALIFORNIA

VIABILITY OF THE CONIDIA OF ENDOCONIDIOPHORA FAGACEARUM BRETZ, IN THE FECAL MATERIAL OF CERTAIN NITIDULIDAE[1]

Frederick F. Jewell

Reports of the transmission of oak wilt by several species of beetles in the family Nitidulidae (2, 4) have established the fact that the beetles are capable of disseminating the spores of the fungus and inoculating trees through fresh wounds. In the absence of information to the contrary, it has been assumed that this was crude mechanical transmission (4). Recently, Bart and Griswold (1) demonstrated the viability of the oak wilt fungus conidia in the feces of Drosophila melanogaster Meigen. They also observed numerous endoconidia in the feces of nitidulid beetles, but did not demonstrate the viability of these spores.

The author also has observed endoconidia in the fecal pellets of nitidulids, but efforts to isolate the fungus from the pellets did not meet with success. When the pellets were plated on agar of various types, including water agar, the spores were quickly overrun by bacteria and yeasts. Because of the difficulty of positively identifying the oak wilt organism or demonstrating the viability of the spores by this technique, other means were sought.

Leach, et al (3), reported the ability of nitidulid beetles to act as agents of spermatization by carrying spores from a mycelial mat of one compatibility type to a mat of the opposite type. Fertile perithecia were produced as a result of this spermatization. It occurred to the author that by using the feces of nitidulids for spermatizing the receptive mycelium, the presence of viable spores could be demonstrated. If fertile perithecia developed as a result of this treatment, it could be concluded that viable spores were present in the fecal pellets used. It was thought that by using this technique the contaminants would not have an opportunity to overrun the conidia as they do on agar. Experiments to determine this were carried out as follows:

Nitidulid beetles, Glischrochilus fasciatus (Ol.) and G. sanguinolentus (Ol.), were fed on type A cultures of the oak wilt fungus for one week. Fresh type A cultures were supplied every two days. The beetles were removed from the plates with flamed forceps and glued upside down on alcohol-washed glass slides placed in a sterile, empty Petri dish. The beetles were then thoroughly brushed several times with a camel's hair brush soaked in 95% alcohol to eliminate surface contaminants. The beetles were then examined under a stereoscopic microscope as defecation occurred, and the pellets were collected immediately from the beetles using a flamed needle. The pellets were then placed in a small drop of sterile distilled water that had been dropped onto the mycelium of fungus colonies of type B, which were seven days old and growing on maltose-Casimino Acids agar. Three plates of the type-A fungus were used as checks. One pellet was placed on each colony on the plates. The plates were then placed under a stereoscopic microscope and the pellets were crushed and dispersed in the drop of water with a flamed

Table 1. Results of spermatizing type A and type B isolates of Endoconidiophora fagacearum with fecal pellets of nitidulid beetles that had fed on type A cultures.

Series	Species of beetle used	Number of pellets tested		Percentage of tested pellets inducing fertile perithecia	
		On Type A	On Type B	On Type A	On Type B
I	Glischrochilus fasciatus	3	15	0	100
II	G. sanguinolentus	0	2	-	100
	G. fasciatus	3	21	0	52
III	G. fasciatus	3	28	0	57

[1] Published with the approval of the director of the West Virginia Agricultural Experiment Station as Scientific Paper No. 476.

needle. The suspension was spread over a portion of the colony with the needle. The needle was flamed after use on each colony. The cultures were incubated at 20° C. for three days and then examined for presence of fertile perithecia.

Three separate series of tests were made. A total of 75 colonies were spermatized with the feces collected from 45 nitidulids previously fed on A type cultures; 66 of the spermatized colonies were type B and 9 were type A. Of the 66 type B colonies 67 % produced fertile perithecia and 33 % produced no perithecia. The number of perithecia produced varied from 2 to 30, mostly 15 to 20, per colony.

The results given in Table 1 clearly demonstrate that conidia of the oak wilt fungus may pass through the intestinal tract of the nitidulid beetles in a viable condition. In all three series, fertile perithecia were produced on a majority of the colonies spermatized with nitidulid feces. The results indicate also that spores surviving in the feces of the beetles could possibly serve as inoculum and cause infection if deposited on suitable fresh wounds, where the living oak tree would serve as a differential medium. Further experimens are under way to determine this definitely. The techniques used in these experiments may be adapted to other studies for determining the presence of viable spores of the oak wilt fungus.

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WEST VIRGINIA AGRICULTURAL EXPERIMENT STATION

FUNGICIDE-INSECTICIDE MIXTURES IN PRE-PLANTING CORM TREATMENTS FOR CONTROL OF BACTERIAL SCAB OF GLADIOLUS[1]

Roy A. Young

Bacterial scab, caused by Pseudomonas marginata (McCull.) Stapp, is one of the most common diseases of gladiolus. Although the productiveness of corms is not greatly reduced by the presence of scab lesions, infected corms are unsightly, hence have less sale value than comparable clean corms. Control of scab has been attempted primarily through use of various types of pre-planting corm treatments. Such treatments must also be effective against corm diseases caused by Fusarium sp. and Stromatinia gladioli (Drayton) Whetzel. Mercurial treatments have been used most commonly to control bacterial scab and other diseases of gladiolus. In general, long treatments such as a 12-hour (2, 3) or one-half-hour (1) soak in mercuric chloride, shorter soaks in mercurous choride (3), or soil treatment with mercurous chloride (3), have given most effective control of bacterial scab. Unfortunately, such treatments have usually retarded germination, growth and blooming, reduced the total yield of corms (3), and have not always given the desired degree of scab control. In Oregon a 30-second dip treatment in New Improved Ceresan, used at the rate of 2 lbs. in 100 gallons of water, gives as good control of Fusarium rot as the more commonly used 15-minute soak and does not have any deleterious effect on growth of gladiolus (4). However, this treatment is not effective in controlling bacterial scab.

The occurrence of bacterial scab in Oregon gladiolus plantings has been difficult to predict. Scabby corms planted in one area may yield clean corms and in another area may yield heavily scabbed corms. Similarly, scab-free corms may yield heavily scabbed corms from some soil and scab-free corms from other soil, irrespective of pre-planting treatment used. The type of soil appears to have little effect on scab development since severe scab has been observed on both heavy and light soils.

The frequent occurrence of scabby corms in soil infested with wireworms suggested that these or other insects might influence the amount of bacterial infection by providing infection courts. If this were true, the addition of an insecticide to the preplanting treatment mixture might reduce the incidence of scab by killing or repelling soil insects capable of wounding the surface of corms and providing points of entry for P. marginata.

To test this possibility, a preliminary trial was conducted in 1951 in which moderately scabby corms were dip-treated in New Improved Ceresan (2 lbs.-100 gal.), New Improved Ceresan plus 2 lbs. of 20% aldrin, New Improved Ceresan plus 2 lbs. of 20% lindane, or received no treatment prior to planting. The mean percentage of scabby corms that subsequently developed from each treated lot was 81.6, 32.0, 33.3, and 87.0 respectively. The incidence of scab on the corms grown from stock treated with either aldrin or lindane was approximately 50 percent less than on corms from stock treated with New Improved Ceresan alone or from untreated planting stock.

In 1952, a larger trial was carried out in which four New Improved Ceresan-insecticide combinations were tested. Six lots of 400, size four, Snow Princess corms were each given one of six treatments, then planted in a random block planting in four replications of 100 corms each in soil where Fusarium rot had not been prevalent. The materials used, and their effect on stand, number of corms harvested, corm weight, and incidence of scab are shown in Table 1.

None of the treatments significantly affected stand, number of corms harvested or weight of corms. However, treatment of corms in mixtures containing 2 pounds of New Improved Ceresan plus 2 pounds of aldrin or 2 pounds of heptachlor per 100 gallons resulted in significant reduction in the incidence of scab lesions as compared to both the New Improved Ceresan treated and the untreated lots. Similar results were obtained in experimental plots in two other areas. The discrepancy in results with lindane in 1951 and 1952 is probably due to the use in 1952 of a concentration too low to be effective.

The addition of insecticidal chemicals to gladiolus pre-planting treatment mixtures should be investigated in other areas. Tests should be conducted also to determine the effectiveness of pre-planting corm treatments as a means of preventing wireworm injury to gladiolus. If effective, treatment of corms would offer a very economical means of wireworm control.

The present recommendation for pre-planting treatment of gladiolus corms for scab control

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Table 1. The effect of pre-planting treatment of gladiolus corms in fungicide-insecticide mixtures on stand, yield and incidence of scab.

Treatment	Stand	No. of corms	Weight of corms	Corms with scab lesions
			oz.	%
NIC[a]	99.2	94.5	94.0	58.3
NIC + 20% aldrin (2 lbs-100 gal.)	98.7	94.2	95.5	24.2[b]
NIC + 20% aldrin (1 lb-100 gal.)	95.5	96.5	92.7	54.1
NIC + 20% heptachlor (2 lbs-100 gal.)	97.7	95.7	95.5	17.6[b]
NIC + 5% lindane (10 ml/gal.)	99.9	94.7	98.2	56.5
Untreated check	97.5	96.0	95.0	71.9

[a] NIC - New Improved Ceresan (2 lbs. in 100 gallons of water). All treatments contained DuPont Spreader-Sticker, 1/2 pt. in 100 gal.
[b] Differences significant at 1% level. Differences necessary for significance are not indicated since percentages were transformed for analysis by arc-sin transformation.

in Oregon is a 30-second dip-treatment in a mixture of 2 pounds of New Improved Ceresan, 2 pounds of 20% aldrin or heptachlor, and 1/2 pint of Triton 1956B or DuPont Spreader-Sticker in 100 gallons of water. This recommendation is based on experimental trials in 1951 and 1952 and observations of successful scab control in a commerical planting in 1953. In three experimental trials, the addition of aldrin and heptachlor to corm treatment mixtures resulted in significant decreases in the incidence of scab and had no deleterious effect on gladiolus that could be measured by emergence, flowering or yield comparisons.

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DEPARTMENT OF BOTANY AND PLANT PATHOLOGY, OREGON AGRICULTURAL EXPERIMENT STATION, CORVALLIS, OREGON

NEW OR UNUSUAL RECORDS AND OTHER BRIEF NOTES ON PLANT DISEASES

1953 EPIPHYTOTIC OF CURLY TOP OF SUGAR BEETS IN KANSAS

By W. H. Sill, Jr., and S. M. Pady

Curly top of sugar beets has been seen in Kansas prior to this year but has been of little, if any, economic significance. This year a severe epiphytotic was reported in the Garden City-Scott City irrigated growing areas. A visit to the area confirmed the report.

Typical symptoms were seen in all fields examined. These were yellowing and dying of older leaves, rolling, curling and puckering of the younger leaves with some slight mottling, phloem necrosis of the roots and petioles, excessive adventitious bud and hairy root development, and much yield reduction owing to reduced stand and small beets. Tonnage was reduced approximately 50 percent per acre but percentage of plant infection was higher, possibly 75 to 80 percent or more. Curly-top resistant varieties were not being grown in the area. This, of course, increased the losses considerably.

Eutettix (Circulifer) tenellus Baker, the vector, was found in some abundance at different times in the area by state and federal entomologists at the Branch Experiment Station at Garden City. Viruliferous leafhoppers apparently were blown in from the southwest during the spring high winds or else possibly picked up the virus enroute from some of the many known available natural virus hosts.

Resistant varieties will be grown next year, and some needed local research on the problem will be initiated.

DEPARTMENT OF BOTANY AND PLANT PATHOLOGY, KANSAS STATE COLLEGE, MANHATTAN, KANSAS

STEM RUST OF MERION BLUEGRASS IN KANSAS

By Clark T. Rogerson and C. L. King

Merion bluegrass in a lawn at Manhattan, Kansas, was found to be heavily infected with stem rust (Puccinia graminis Pers.) on October 22, 1953. Only the uredial stage of the rust was present.

This lawn was planted in the fall of 1952 and made an excellent stand. The following spring the grass was in very good condition but during the summer dead spots began to appear and considerable thinning of the stand occurred. The lawn was watered frequently throughout the season. Isolations from the dying plants revealed the presence of Helminthosporium sativum Pam., King & Bakke. Hence the trouble was diagnosed as the melting-out disease of bluegrass.

Stem rust was most noticeable on the grass in the thinned areas of the lawn, these plants being conspicuously yellow in color. Inspection revealed that the rust was present on plants throughout the entire lawn although more uredia per leaf were evident on the uncrowded plants. Nearby lawns of ordinary Kentucky bluegrass showed no evidence of the rust.

Because of the increasing interest in Merion bluegrass, it seems desirable to record its susceptibility to stem rust. Ray (PDR 37: 578, 1953) has reported that Merion bluegrass is also very susceptible to leaf rust (Puccinia rubigo-vera (DC) Wint.).

DEPARTMENT OF BOTANY AND PLANT PATHOLOGY, KANSAS STATE COLLEGE, MANHATTAN, KANSAS

NEW LOCATIONS FOR FUSARIUM WILT OF SESAME

By. G. M. Armstrong

I wish to report two new locations for the Fusarium wilt of sesame. This summer, the county agent at Savannah, Georgia, sent diseased plants from which we isolated a Fusarium. A pathogenicity test has just been completed which shows that this is the sesame wilt fungus. A new location for the sesame breeding work at Clemson was established last summer, 1952. In August 1953, the second season the plants were grown on this site, wilting plants were called to my attention. Recent inoculation tests in the greenhouse have shown that this is also the Fusarium wilt disease. The breeding work in sesame is done by Mr. J. W. Martin, Department of Horticulture, and he wondered if the fungus had been introduced with the seed. The pattern of distribution of the disease over the field did not indicate to me that the fungus was introduced with the seed.

CLEMSON COLLEGE, CLEMSON, SOUTH CAROLINA

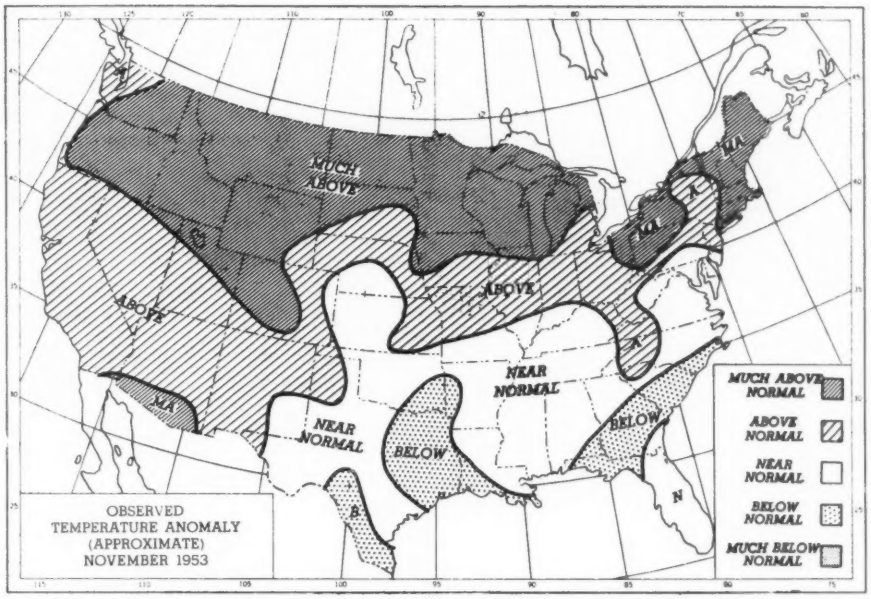
DETRIMENTAL EFFECTS OF COPPER SPRAYS TO NORWAY MAPLE IN RHODE ISLAND

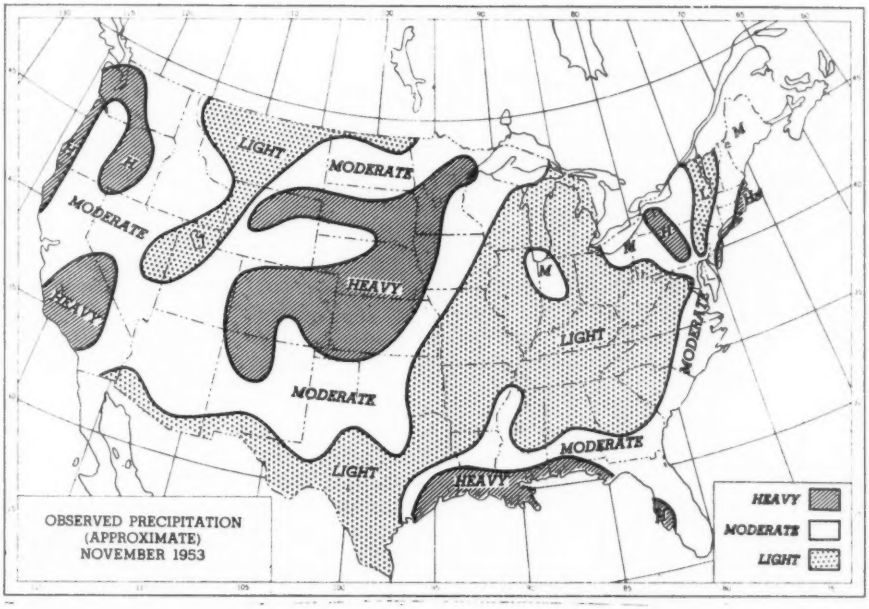
A. C. Tarjan and
F. L. Howard

Two years' tests on the application of copper sprays to foliage of Acer platanoides L. show that marginal and blotch necrosis result on sprayed leaf surfaces. In 1951, Bordeaux mixture applied June 11 and July 2 at the rate of 8-4-100 to trees growing in nursery rows resulted in a severe marginal leaf necrosis involving from 20 to 50 percent of the leaf area. In 1952, two applications of Bordeaux applied two weeks apart in June and at the same rate as in 1951 caused similar symptoms of injury. Undersides of leaves from sprayed trees exhibited a blackish russeting consisting of numerous minute flecks which coalesced to give a blotched appearance. In some cases brown necrotic lesions were present within the russeted areas. An average of 36 percent of the leaf surfaces was found to be affected.

Robertson copper applied at the rate of 3 lbs. per 100 gallons of water in 1951 caused a blotching and marginal necrosis of from 5 to 25 percent of the leaf areas. In 1952, this chemical applied at the same rate injured an average of 14 percent of the leaf surfaces. Therefore, contrary to some published recommendations, certain copper fungicides are likely to cause injury to Norway maple foliage.

RHODE ISLAND AGRICULTURAL EXPERIMENT STATION, KINGSTON





The terms used in the accompanying maps, which define the ranges of temperature and precipitation, are numerical class limits. These are based on a statistical analysis of past records through which is determined the normal frequency of occurrence of temperatures and precipitation at various times of the year for different locations. For temperature the classes above, below, and near normal are so defined that they each normally occur one-fourth of the time; much above and much below normal, one-eighth of the time. Precipitation is depicted in terms of light, moderate, and heavy, each class normally occurring one-third of the time and thereby having equal probability of occurrence. These maps graphically represent only the general trends and give the country's weather picture at a glance. For quantitative studies, where monthly mean temperatures and actual precipitation records are needed for a given time and place, other publications of the Weather Bureau should be consulted. P. R. M.